EVALUATION OF PRICK TEST IN VARIOUS ALLERGIC DISORDERS

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C E R T I F I C A T E

This is to certify that the work entitled "EVALUATION OF PRICK TEST IN VARIOUS ALLERGIC DISORDERS" has been conducted by VEER MAHENDRA PAL SINGH under my direct guidance and supervision in the department of Pediatrics, M.L.B. Medical College, Hospital, Jhansi.

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17/3/93

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(Veer Mahendra Pal Singh)

C O N T E N T

INTRODUCTION

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The term 'allergy' was coined by Von Pirquet in 1906 to include two different aspects of immune response to allergen (antigen) challenge : the beneficial response called immunity and harmful one called hypersensitivity.

Allergy is most commonly used as synonym for hypersensitivity but some times employed in a narrow sense to refer to only one type of hypersensitivity i.e. atopy. For the induction of hypersensitivity reactions, the host should have prior contact to the same antigen (Allergen).

Ellis (1987) defined allergic disorder as 'Adverse physiological reaction resulting from the interaction of antigen with humoral antibody and/or lymphoid cells. Type-I hypersensitivity, mediated by IgE, is of greatest interest to the allergist.

Allergic diseases (asthma, rhinitis and urticaria) are important causes of morbidity ranging from trivial discomfort to total incapicitation and abstinence from school. Nasobronchial allergy is quite common in India and allergy is considered to be the most important factor in causing bronchial asthma. Causal allergens vary from place to place depending upon local environmental factors. The most common offending agents are inhalants which include dusts, pollens, and fungi etc.. Pollens and

dust allergens comprise 75% of all the respiratory allergens (Shivpuri, 1966). The statistics of allergy in India is incomplete, but Vishvanathan (1964) estimated about 10% population in the country suffers from one or the other allergic disorders. Williams and Mecnicol (1969) in their study found that 3.7% of population had regular episodes of asthma from early childhood to ten years of age. Hence, it is important that offending allergen(s) be identified in each individual and whenever possible appropriate immunotherapy be given to the patient.

Today the skin prick test is widely used as a laboratory procedure of choice to identify offending allergens in respiratory allergy which are IgE mediated. The tests are performed on volar aspect of forearm. Moreover, results of skin prick test are more immediately available (45 minutes as compared to 2-3 days in RASTs). Skin test is more sensitive, faster and relatively less expensive to the patients. Thus, skin testing with particular allergen remains the most revealing procedure in diagnosing specific allergic factor(s)(Michael et al, 1987).

In view of such problems, present study was designed to study the spectrum of allergic illness in children, and to confirm their allergic nature by skin prick test using 21 allergens. An approach was made to know about the family history, absolute eosinophil count,

precipitating factors, and relation of parasites in allergic disorders. The reactivity to histamine at different ages was also noted. Furthermore, the effectiveness of immunotherapy was also observed.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Von Pirquet (1906) coined the term allergy for a 'state of changed reactivity in a host, occurring as a result of contact with a foreign substance. He put together the Greek words "allos" meaning different or changed and 'ergos' meaning work or action. This altered reactivity could either be beneficial to host as in the case of immunity or detrimental as in anaphylaxis. In modern usage allergy refers only to adverse effects.

ONTOCOGY OF IMMUNE SYSTEM

The immune system arises from developing lymphoid tissue during embryogenesis. The lymphoid tissues are of two types i.e. central and peripheral. In our case the central lymphoid organ is the thymus.

In humans B cell (Bursa dependent) are involved in humoral or antibody mediated immunity and T cells (Thymus dependent) involved in cell mediated immunity. Their differentiation occurs in thymus and probably gets stimulated by a humoral factor produced by the thymic epithelium termed as thymopoietin or thymosin(Goldstein et al, 1974)

Hall (1974) proposed that T cells migrate from the thymus by way of blood stream and lymphatics to populate the peripheral lymphoid organs i.e. lymphnodes, spleen, bone marrow, tonsils and gut associated lymphoid system.

Gathings and Cooper (1977) observed that B cells precursors are demonstrable in the mammalian fetal liver and adult bone marrow.

Pearl (1978) opined that migration of B cell to the peripheral lymphoid organs also occurs.

Pierce and Kapp (1978) postulated that a third lymphoid cell evolving from the central stem cell line during this period is the monocyte or in it's mature form macrophage and over the past several years it has been recognised that interaction of macrophage with T and B cells is important in the initiation of immune response and it's regulation.

HUMORAL IMMUNITY

Stimulation of the production of specific antibodies depends upon the nature of antigen. Antibody activity in humans reside in five major classes of globulins. These immunoglobulin classes are termed as IgG, IgM, IgA, IgD and IgE. Each immunoglobulin class appears to be synthesized by a separate B cells subclass. It has been recognised that for maximal B cell IgM and IgG primary responses to occur to most antigens, the presence of T cells is required (Katz et al, 1972). This population of T cell is known as Helper T cells (Friedman and Greaves, 1973).

In contrast to the majority which are T dependent antigens, there are a few antigens which do not

require mediation by T cells. These are called as T independent antigens (Moller, 1973).

Along with the interaction of B and T lymphocytes, macrophages also play a vital role. Initially antigenic proteins bind to macrophage before their recognition by T lymphocytes (Basten et al, 1976).

Benacerraf (1978) suggested that macrophage process or degrade the antigen and then the antigen is combined with a product of genes which are linked to the histocompatibility complex (MHC) of the species. This gene product was called Ia or Immune Response Associated, presumably coded for by a corresponding Ir or Immune Response Gene.

Again Benacerraf and Germain (1978) wrote that the combination of processed antigens and Ia was presented to T cell for immune recognition and stimulation to helper activity or effector activity.

Seligmann and Franklin (1978) suggested that B lymphocytes have readily identifiable IgD and IgM manomer present on their cell membrane.

REGULATION OF ANTIBODY SYNTHESIS

It is known that following the interaction of macrophages, helper T cells and B cells, the B lymphocytes undergo blastogenesis and are transformed into mature plasma cells, which synthesize specific class immunoglobulin. Many regulatory mechanisms, however, operate over this.

IgM B cells are suppressed by circulating IgG antibody directed against the same antigenic specificity (Sercurz et al, 1974).

There is a growing evidence that suppressor T cells exert a regulatory effect and yet another T cell subpopulation may exist, generating an opposing amplifier signal to which, the mature B cells is subjected (Markham et al, 1977).

Thus, amplifier and suppressor T cells appear to generate complementary effects to keep the degree of B cell activity appropriate to antigenic stimulation (Benacerrag, 1978).

Macrophages also exert modulatory influence on B cell biosynthesis mediated by elaboration of so called monokine which enhances antibody formation (Dimitriu and Fauci, 1978).

Populations of suppressor T cells regulate IgA and IgE synthesis also. Initiation of a specific IgE response is dependent on interaction between the macrophage, helper T cells, and IgE B cell analogous to the IgG response.

IMMUNOGLOBULINS

Immunoglobulins are a heterogenous group of proteins belonging to gammaglobulin fraction of the serum proteins. Their main function is to act as antibodies.

The gammaglobulins were first designated by Tiselins in 1937 as a distinct group of serum proteins having a distinct electrophoretic mobility.

Kunkel and Putnam (1953) showed that myeloma proteins in the serum of patients with multiple myeloma belonged to gammaglobulin fraction of serum proteins.

Graber et al (1953) supported the finding of Heidelberger and also separated the newer IgA besides the IgM and IgG by an immunoelectrophoresis technique.

Porter (1959) was able to clear the immunoglobulins into two fragments, separable by ion exchange chromatography; one fragment retained the capability to react with the immunogen and was called Antigen binding fragment or Fab, the other crystallized upon standing and was called crystallizable fragment or Fc.

According to Edelman and Poulik (1961), globulin could be split into two components by reduction with thiols in the presence of urea i.e. the heavy chains (Molecular weight 50000) and light chain (molecular weight 20,000).

Grey and Kunkel (1964) discovered the subclasses of IgG, viz. IgG_1, IgG_2, IgG_3 and IgG_4.

Rowe and Fahey (1965) discovered the fourth class of immunoglobulins viz. IgD from an atypical myeloma protein.

Kunkel and Prendergast (1966) discovered the subclass of IgA.

IgE

In 1967, Ischizaka et al discovered that reagents or skin sensitizing antibody belonged to a unique class of immunoglobulin which they called IgE and it remained as a principals if not the sole mediator, of type I hypersensitivity reaction.

Johansson et al (1970) found that IgE level varied with age but not with sex. Patients with asthma and atopic dermatitis had raised IgE level more often than patients with allergic rhinitis. Atopic dermatitis had the highest mean IgE level.

Tada (1975) found that 1% of the total IgE was cell bound.

In 1975, Norman suggested that IgE was the principal mediator of immediate (type-I) hypersensitivity reactions.

IgE binds to tissue mast cells and basophils. This cell bound complex then causes degranulation of basophils on combining with antigen (allergen), with the result histamine and other mediators are released (Beaven, 1976).

Homburger (1978) suggested that IgE concentration was generally higher in the allergic population, although there was some overlapping with non atopics.

Biological properties of IgE class were investigated and presented by Ishizaka et al (1978).

Zeiss (1980) discovered that Fc fragment of immunoglobulin was responsible for the protein's ability to fix to receptors on mast cells and basophils.

Leung et al in his review (1985) concluded that T lymphocytes played an important role in the isotype specific regulations of the human IgE response.

CELL MEDIATED IMMUNITY

Parker (1976) concluded that the parellel mechanism of immune recognition and immune regulations were analogous in humoral and cell mediated immunity. However, immune reaction in cell mediated immunity was brought about by sensitized lymphocytes, rather than by free antibody molecule.

The development of antigen specific T lymphocyte is dependent on interaction of macrophage and T cell.

Benner and Van Ouden aren et al (1977) showed that memory T cells were long lived and maintained immunologic memory of previously encountered antigens.

Kapp et al (1978) postulated that suppressor T cells were responsible for establishing tolerance to self antigens.

Effector T cells, on contacting with antigen created the molecular, cellular and clinical manifestation of cell mediated immunity reaction (Binz, 1978).

CLASSIFICATION OF HYPERSENSITIVITY REACTIONS

Gell and Coomb (1963) classified the immune response into four types i.e. type I to IV hypersensitivity reactions, of which type I is known as anaphylactic or reagin dependent. Several modifications however have been proposed to the Gell and Coomb classification system (Sell, 1975).

Raitt (1972) added fifth category of immune response i.e. stimulating antibody reaction and Irvine (1984) described the sixth i.e. antibody dependent cell mediated cytotoxicity (ADCC).

TYPE - I ; ANAPHYLACTIC REACTION

This type of reaction is also called immediate type hypersensitivity or reaginic hypersensitivity and is caused by reaginic antibody of the IgE class (IgE_4 in a few cases) e.g, extrinsic bronchial asthma, allergic rhinitis, partly atopic dermatitis, most cases of anaphylactic shock, some cases of urticaria and angic-edema (Lakin and Cahill, 1976).

Antigens which cause type-I reaction are called allergens. When the allergen reacts with IgE attached to the surface of the mast cells, the cell degranulates and liberates chemical mediators, responsible for symptoms.

Main chemical mediator is histamine and others are SRS-A (Leukotriene $C_4+D_4+E_4$), leukotriene B_4 ECh-A (eosinophil chemotactic factor of anaphylaxis, platelet

activating factor (PAF), TxA_2, a high molecular neutrophil chemotactic factor(MCF) and prostaglandin D_2 (Mathe et al, 1977).

In 1978, Lichtenstein concluded that these chemical mediators were responsible for vasodilation, increased capillary permeability, and smooth muscles contraction, which were manifested clinically as urticaria angio-edema, hypotension, bronchospasm, spasm of gastro-intestinal musculature, or uterine contractions, depending on the location and severity of the reaction.

In contrast to other cell types, mast cell and basophils have high affinity receptors for IgE, these are about 10^5 per cell.

TYPE-II : CYTOTOXIC REACTION

This reaction is also termed as complement dependent cytotoxicity. Complement system also mediates type III or toxic complex reaction.

TYPE-III : TOXIC COMPLEX REACTION OR IMMUNE COMPLEX REACTION

In this reaction, complexes are formed between circulating antigen and specific antibody, especially of IgE class. In this reaction complement system is also activated, causing local infiltration by neutrophils, which in turn release tissue damaging enzymes.

Dixon (1965) concluded that arthus reaction experimental and clinical serum sickness, few glomerulo-

nephritis and some drug reactions, followed the mechanism of type III reaction.

Fink and Salvaggio (1978) thought that type III reaction played a significant role in the pathogenesis of various hypersensitivity pneumonias.

TYPE - IV : CELLULAR HYPERSENSITIVITY REACTION

This is also called delayed hypersensitivity reaction, as a delay of 24 to 72 hours occurs in the initiation of reaction and is mediated by antigen specific sensitized T lymphocytes. Sensitized T lymphocytes also act by liberating lymphokines, which mobilize non sensitized cells to fight the antigen.

David et al (1964) observed as little as 2.5% antigen specific T lymphocytes to the total cell population, in the delayed reaction.

TYPE - V : STIMULATING ANTIBODY REACTION

This reaction was considered as a modification of the type II reaction, by Raitt (1972), in that the specific antibody combines with antigen on the cell surface but complement was not activated.

The classical example of this reaction is in Graves' disease (Exophthalmic Goitre) in which excess amounts of thyroid hormones are produced.

TYPE - VI : ANTIBODY DEPENDENT CELL MEDIATE CYTOTOXICITY(ADCC)

K cell mediated cytotoxic mechanisms may be important in the pathogenesis of auto-immune disease and

in tumour rejection. K cells were also involved in the defence against helminthic infections such as schistosomiasis where the size of the parasite is too large for effective phagocytosis.

DIAGNOSIS OF ALLERGY AND HYPER-REACTIVITY

It was earlier believed that allergy was the cause of almost all cases of asthma, rhinitis and dermatitis. When an allergen could not be identified, bacterium, food, or yet unidentified substance was incriminated, However, it has been realized that increased non-specific responsiveness of the diseased organ, hyper-reactivity, is of considerable importance (Cockroft, 1983).

For the diagnosis of allergy and hyper-reactivity, following points are considered (Mygind Niels, 1986) :

1. History
2. Physical examination
3. Exercise test
4. Histamine test.
5. Blood eosinophil count
6. Total serum IgE
7. Skin testing with appropriate allergen.
8. RAST
9. Bronchial allergen provocation test.

In 1921 Prausnitz and Kustner for the first time demonstrated transfer of immediate hypersensitivity from an affected individual to a normal individual through serum.

Atopy and atopic refer to certain allergic diseases which have a familial tendency to occur and are associated with eosinophilia of blood and tissue secretions (Coca, 1923).

In 1925, Coca and Grove did extensive studies of skin sensitizing factors from the sera of the patients with ragweed hay fever.

The cutaneous reactivity of infants and neonates is reduced (Sulzberger et al, 1940; Matheson et al, 1954 and Kaufman, 1971).

Herzheimer et al (1954) studied and evaluated the role of skin test in respiratory allergy.

Chambers et al (1958) showed that asymptomatic subjects, who are skin test positive (ragweed pollenosis) are at a higher risk of developing an allergic syndrome, if less than the 40 years of age, as compared to subjects who are skin test negative.

Curran and Goldman (1961) found 50% of the non-allergic individuals with a positive family history of atopy and positive skin tests to aeroallergens, compared to only 9% of subjects with a negative family history.

In 1965 Reinberg et al demonstrated that skin reactivity to histamine and to compound 48/80 was maximal between 7 to 11 PM and was at its nadir et 7 AM.

Morrow Brown et al (1968) studied the role of mites in house dust, by skin testing. They found that dermatophagoides, pteronyssinus played an important role in sensitizing susceptible people. Hence the use of mite extract was considered to be an important advance in the diagnosis of allergy to house dust. They also found in their study that in 50% cases, there was no history to

suggest house dust sensitivity, especially in children, but nasal provocation tests were all positive.

Morrison Smith et al (1969) in their study, 'clinical significance of skin reactions to mite extract in children with asthma found that more than 50% of school children in Birmingham suffering from asthma showed positive prick test reactions to house dust, but these reactions were small and relatively less frequent than prick test reactions to gross pollen in children with pollen allergy. Though positive reactions to D. pteronyssinus were obtained more frequently and were of greater size than those from other extracts, it was considered that D. farinae was a suitable substitute for D. pteronyssinus for skin testing.

Hagy and Sethipane (1969) in their study showed that skin test reactivity in the range of 20 to 49% was present in the general population.

Louse and Lubs (1971) concluded that allergic diseases had both environmental and hereditary factors operating but environmental factors were predominant. This was shown by low concordance in the monozygotic twin group (25.3%) and in the dizygotic group (16.1%).

Study of Lal et al (1973) showed that there was a high incidence of positive skin reactions to extracts of both mites and house dust in individuals with extrinsic bronchial asthma. These reactions showed a significant correlation with the clinical history of house dust

allergy and bronchial asthma. The presence of mites in 19 out of the 25 samples analysed and positive skin test clearly suggested that the house dust mite of dermatophagoides species was present in this part of the world and was playing a significant role in the etiopathogenesis of bronchial asthma.

Gleich et al (1974) noted the highest allergen specific IgE levels between 12 and 20 years of age.

Hendrick et al (1975) had reported decreased prevalence of skin reactivity with age. Peak skin test reactivity has previously been reported to be the highest in 15 to 30 years old age group (Pearson, 1987).

Barbee et al (1976) believed that sensitivity of skin tests was decreased in the elderly.

Godfrey et al (1976) evaluated the prevalence of immediate positive skin test to D.pteronyssinus and grass pollen in school children. The range of prevalence for positive skin tests in allergic population was 50 to 95%.

Lee et al (1977) confirmed the observations to skin test responses to grass and house dust extracts. They suggested that false negative readings could rerult if skin tests were performed in the early morning office hours.

Cavanaugh et al (1977) evaluated the clinical values of bronchial provocation testing in childhood asthma.

Skin test reactivity was positively correlated with total IgE and specific serum IgE levels (Pascual and Reddy et al, 1977).

In 1978, Reddy et al, made a re-appraisal of intercutaneous tests in the diagnosis of reaginic allergy.

Skin test reactivity to an allergen was also highly correlated to both basophil sensitivity and tissue (bronchial nasal)sensitivity to that allergen (Cockcroft et al, 1979).

Brown et al (1979) studied the relationship of skin test reactivity and serum IgE in cases of respiratory allergy.

Skin tests were used to diagnose allergic disorders in infancy (Businco et al, 1979; and Warner, 1980).

Prick tests are totally harmless in infancy and the reproducibility may be improved by the use of the recently standardized devices (Nelson, 1983).

The skin prick test is more sensitive than the scratch test. Intradermal skin testing is higher in sensitivity but less specific than epicutaneous skin test. Thus, a greater number of false positive reactions can occur in intradermal skin testing. In circumsrances where intradermal tests may be dangerous (certain foods and drug allergies), skin prick testing is particularly useful as the initial form of allergy testing (Krouse et al, 1980; Brown et al, 1981; Robert et al, 1983).

Ellis (1983) defined the role of complement in atopic diseases.

According to Position, Statement of the Practice Standards Committee (1983), the skin tests appear to be superior to currently available RASTS in the diagnosis of certain life threatening anaphylactic states in which maximum sensitivity is important, particularly in the diagnosis of penicillin and Hymenoptera allergy. The results of skin tests are more immediately available. Where both tests can be initiated at the time of the patient's visit the result of skin tests are available in about 45 minutes, those of RASTS are available in 2 to 3 days. RAST is preferable to skin testing in certain conditions where skin testing is unsatisfactory, particularly where there is dermatographia or widespread skin disease.

Allen D. Adinoff et al (1983) observed that skin testing was standard clinical method for demonstrating the presence of allergen specific IgE antibody in allergic diseases.

According to Anderson (1984) non-immunologic mechanisms also played a role in the pathogenesis of atopic disease.

There was a good correlation between IgE, FAST test, RAST and skin testing (Tsay et al, 1984; and Mirone et al, 1987).

According to Menardo et al (1985) skin test reactivity to histamine and mast cell degranulating agents was lower in newborn infants as compared to adult.

Skin tests represented a major tool in the diagnosis of immediate type allergy (Skassa-Brociek et al, 1985).

Diminished end organ responsiveness in infants and elderly individuals to inflammatory mediators appear to be one contributory mechanism for decreased prevalence of allergen skin test reactivity at extremes of age (Van Asperen et al, 1985).

Jean Luc Menardo et al (1985) studied and confirmed that prick tests could be performed and interpreted without difficulty in infants, keeping in mind that small wheal size was produced by both positive control solutions and allergens.

Rosario Scolozzi et al (1987) believed that presently no in-vitro technique was as sensitive as skin test for allergen specific diagnosis of inhalant allergic disease.

Nalebuff et al (1989) suggested that skin test was more sensitive, faster and relatively less expensive in comparison to RAST.

Pakit Vichyanond et al (1989) observed in their study, that there was no difference in wheal and erythema sizes between morning versus evening skin testing.

Hattevig et al (1989) noted a decreased incidence and severity of atopic dermatitis in first 6 months of life, if the lactating mothers avoided eggs, cow's milk and fish during the first 3 months of breast feeding.

SPECTRUM OF ILLNESS : BRONCHIAL ASTHMA

Asthma represents most serious but common allergic condition of childhood. The term asthma is derived from Greek word meaning "Struggling for breath". There is no universally accepted definition of asthma.

Bruce (1958) observed that nearly 50% patients of asthma had family history of allergy.

In 1964, Vishwanath defined that bronchial asthma is a syndrome that is characterised by attack of expiratory dysponoea, not attributable to disease of the heart or the lung. The smooth muscle in the bronchi and bronchioles exhibits spasm, edema and exudation following exercise, natural exposure to strong odour, irritant, fumes, tobacco, smoke, cold air, intensional exposure to parasympathomimetic agents.

Bronchial asthma may be regarded as diffuse obstructive lung disease with hyper-reactivity of the airways to variety of stimuli and high degree of reversibility of obstructive process which may occur either spontaneously or as a result of treatment.

In 1964, Rockeman divided asthma into extrinsic asthma and intrinsic asthma. Differences were described as follows :

	Extrinsic	Intrinsic
Age of onset	3-35 years	⁄3 and 735 years
Symptoms	Seasonal and perenial	Increased in winter, increased by cold air, infection, pollution.
Mucus	Clear & foamy	Thick and white or colourless
Atopy	Positive	Absent
Skin test	Positive	Negative or positive-non-correlation
Serum IgE	High	Normal
Response to therapy	Good response to bronchodilator and immunotherapy	Poor response to bronchodilator, no response to immuno-therapy.

Eosinophilia was reported in majority of cases of bronchial asthma by Lowell et al (1967) and Sharma et al, (1974).

Samter and Beers (1968) described a special category of non-antigenic asthma, which was induced by the ingestion of aspirin. Skin test to aspirin was always negative in these patients.

Szentivanyi's theory (1968) considered asthma to be due to abnormal beta adrenergic receptor, adenylate cyclase function with decreased adrenergic responsiveness.

Willium and Mecnicol (1969) in his study found that 3.7% population had regular episodes of asthma from early childhood to 10 years of age. In adult the incidence of asthma was 1% of population, approximately.

Berg et al (1969) observed high serum IgE level in 90% cases of perenial asthma and 50% cases of seasonal asthma.

Gleich et al (1970) also observed high serum IgE level in allergic asthma.

Jarrett (1972) suggested that potentiation, as a result of parasitic infestation, could increase the severity of an individual's allergy by elevating specific IgE level to undesired allergens, such as ragweed pollen etc.

In 1973, Robinson suggested that the asthmatic paraxysm is triggered by the hypersensitivity reaction or by mental stress, Allergy was always a basic factor of an asthmatic paraxysm.

Willium and Micol (1973) observed that 90-95% of asthmatic children had atopic constitution to develop type I hypersensitivity demonstrated by skin prick testing by various allergens.

In 1973, Jarrett showed in his study that some allergic children gave positive skin test for thread worm antigen and so the author suggested that hypersensitivity to E. vermicularis allergen absorbed from the bowel might contributes to the allergic signs and symptoms. There was no effect of treatment.

Parker and Smith (1973) thought that the decreased beta adrenergic receptor on leukocyte, of non adrenergic

drug treated asthmatics may provide the morphological basis for the observed hyporesponsiveness to beta agonist.

Alternatively increased cholinergic activity in the airways had been proposed as fundamental defect in asthma, perhaps due to some intrinsic or acquired abnormality in irritant receptors which seem in asthmatics to have lower threshold for response to stimulation (Nadel, 1977).

Gupta et al (1975) observed eosinophilia irrespective of the type of asthma in their study.

Lee and associates (1976) observed that about 11% of population have asthma by 3 years of age.

Agrawal et al (1979) reported significantly high absolute eosinophil count (917.71±618.9) in cases of bronchial asthma compared to control (231.4±105.4).

A role of viral infections in the allergic sensitization process has been postulated by Frick, German and Mills (1979) who studied 13 children with 2 allergic parents, 11 developed clinical allergy and five developed asthma after an attack of respiratory viral infection.

Eosinophil in intestinal parasitic infestation not only involved quantitative change in eosinophil numbers but also a qualitative change in functional capacity that rendered circulating eosinophils more efficient in resisting parasitic infestation. David et al (1980) proved this by showing increased capacity of eosinophils to kill schistosoma mansoni larva in vitro.

Sims et al (1981) observed that about 5% of children suffered from frequent wheezy episodes at some time in their childhood and incidence increased to 20% if children having less than 6 attacks of wheezing were included. It was also observed that about half of the children presenting with asthma were atopic and associated with common minor immunodeficiencies. This disease started at any age; about 80 to 90% of asthmatic children had their first attack before the age of 5 years.

Lukza et al (1982) found eosinopenia in case of acute asthma and eosinophilia in cases of chronic and stable asthma.

Eosinophilia in varying degree, 30 to 40% was also reported by Archarya (1963) in their study of childhood asthma.

Prior to puberty about twice as many boys as girls were affected, thereafter the incidence of sex was equal (Ellis, 1983).

Paihi et al (1990) showed agreement between a history of asthma with allergy to house dust and bronchial challange to whole house dust. All allergic patients had significant bronchoconstriction whereas no reaction could be elicited in the non-allergic group.

ALLERGIC RHINITIS

Seasonal allergic rhinitis is a symptom complex seen in children who have become sensitised to wind borne

pollen of trees, grasses and weeds. It is characterized by watery rhinorrhoea, nasal congestion, sneezing and itching of eyes, nose and throat.

Connell (1969) defined that there may occur inflammation following the acute phase reaction due to hyper reactivity of allergic nose to a variety of non-specific stimuli such as cigarette smoke, strong oddurs, air pollution and climatic changes.

Phillips has shown that an individual requires two more seasons of exposure before exhibiting clinical manifestation of disease.

Cell bound IgE antibodies in response to antigenic stimulation, cause release of mediators, immune reaction and bring about manifestations of the disease (Kaliner et al, 1973).

Incidence of allergic rhinitis was reported to be 10% in general population by Viner and Jackman (1976).

Peripheral blood eosinophils of 4-8% may or may not be present in active allergic rhinitis, but characteristic eosinophils of the nasal secretions obtained during the period of symptoms may be of diagnostic value (Tennenbaun et al, 1980).

From 3-10%, patients of allergic rhinitis could develop asthma or other atopic diseases (Ellis, 1983).

According to Michael Kaliner (1987) skin testing with potent antigenic preparations and positive and

negative control substances remains the most revealing procedure in diagnosing specific allergic factors associated with allergic rhinitis.

URTICARIA

It consist of raised erythematous skin lesions, which are marked by pruritis.

Angioedema is characterised by asymmetrical swelling of tissue. This is like urticaria but involves deeper tissue. Urticaria and angioedema may occur together.

Triple response observed by Lewis (1927) consist of erythema due to capillary and vascular dilatation, edema due to increased capillary permeability and flare due to axon reflex. Intercutaneous injection of histamine inflicts similar type of response along with pruritis implicating that histamine mediates the urticarial response.

Temperature changes induce urticaria with considerable frequency. Idiopathic acquired cold urticaria is probably the most common example of this(Anderson, 1967).

Pasricha (1972) undertook the study to ascertain how far gastrointestinal parasites were responsible for producing urticaria.

Mathews (1974) concluded that nearly 20% population at some time in life suffer from some form of urticaria.

Acute urticaria persists less than 6 weeks while episodes which lasts more than 6-8 weeks are referred as

chronic urticaria. Pathogenesis is mediated by histamine release.

Habte Gabr et al (1976) in his preliminary study showed an important association between chronic urticaria and intestinal parasites.

In urticaria pigmentosa, wheals occur in areas of trauma to the cutaneous benign mast cells tumours which characterize this disease. These pigmented macules or papules are seen most often in childhood, and the diagnosis is confirmed, if rubbing the lesions which produce urtication (Darier's sign).

FOOD ALLERGY

Dees (1959) reported incidence of food allergy in children as 3%.

Fries (1959) after his study concluded that incidence of food allergy decreased with the advancing age.

According to Golbert (1969) food allergy cause a variety of cutaneous, gastrointestinal and respiratory manifestations; urticaria and angioedema are the most common.

The clinical manifestation of food allergy usually result from type I hypersensitivity (Golbert, 1970).

Chua et al (1976) have shown that positive cutaneous tests neither establish nor confirm a definite diagnosis of clinically significant food allergy.

May (1976) also has similar opinion.

Both Chua et al and May demonstrated presence of reaginic antibodies in patients who had negative prick test.

EOSINOPHILIA AND ALLERGY

Eosinophil leukocyte is characterised by the presence of large coarse cytoplasmic granules of prominent red colour (Romanowsky staining method) and by a nucleus which has one or two segments.

Apart from phagocytic and cytotoxic activity eosinophils are attracted to the site of immediate hypersensitivity reaction and has the unique potential to modify and regulate the reaction.

Eosinophils normally account for fewer than 5% of circulating leukocytes. Eosinophils counts more than 5% in peripheral smear or 250% cells per cmm is considered elevated. Blood eosinphils in the allergic disorder does not exceeds 15-20%, but may occasionally be high as 33% in allergic conditions.

In asthma eosinophils play dual part in protecting the patient from the effects of mast cell vasoactive mediators and simultaneously damaging the bronchial mucosa.

Bray and Smith (1931) found that eosinophilia was predominantly associated with allergic disorders.

Eosinophilia was reported in majority of cases of bronchial asthma by Lowell et al (1967).

Sehgal et al (1973) in a study of 158 patients with urticaria and angioedema reported eosinophil count of more than 10% in 26.6% cases.

Gupta et al (1975) observed eosinophilia irrespective of the type of asthma in their study.

Agrawal et al (1979) also reported significantly high absolute eosinophil count (917.71±618.9) in cases of bronchial asthma, compared to controls(231.4±105.4).

Lukza et al (1982) on the other hand found eosinopenia in cases of acute asthma and eosinophilia in cases of chronic and stable asthma.

Eosinophilia in varying degree, 10 to 40% was also reported by Acharya (1983) in their study of childhood asthma.

In addition to atopic illnesses and parasitic infections many infectious, inflammatory, neoplastic and even immunodeficiency problems are associated with profound alteration in circulating eosinophils, thus limiting the diagnostic significance of eosinophilia.

PARASITES AND ALLERGY

Jarrett (1972) have suggested that potentiation as a result of parasitic infestation, could increase the severity of an individuals allergy by elevating specific IgE level to undesired allergens, such as ragweed pollen etc.

Pasricha (1972) underlook the study to ascertain how far gastrointestinal parasites were responsible for

producing urticaria. Incidence of parasites in urticaria was not different from that in other dermatological diseases (61.5% and 72% respectively). Twenty five patients of urticaria harbouring gastrointestinal parasites were treated but only in two patients was the elimination of E. histolytica associated with a significant decrease in the intensity of urticaria.

Habte Gabr et al (1976) in their preliminary study showed an important association between chronic urticaria and intestinal parasites. Urticaria in 11 out of 14 patients had direct relationship to intestinal parasites, particularly Ascaris and was cured when the specific parasites were eliminated with anti-helminthic drugs.

Pasricha et al (1979) again surveyed the causes of urticaria and found only 7(1.4%) cases in whom elimination of parasites had resulted in relief from urticaria. Their studies however, did not account for urticaria caused by allergy to Larva of Ascaris and Hook worm as they traversed tissues before reaching gastrointestinal tract. Such cases of urticaria were likely to be of short duration as once larvae reached gastro-intestinal tract and matured, the antigenic stimulation would disappear, as opined by authors.

Veronesi et al (1982) established a relationship between intestinal giardiasis and urticaria. They found in their study of 50 patients of chronic urticaria,

giardia in stool and all of them improved with metronidazole, which was more than coincidental.

Hamriek et al (1983) also reported cases of urticaria caused by giardial infestation. They reported that urticaria could occur after massive absorption of antigen following the death of parasite.

Twarog (1983) opined that parasitic infestations should be considered in individuals having urticaria specially in those who came from an endemic area, had peripheral eosinophilia and who had elevated IgE.

IMMUNOTHERAPY

The concept of immunotherapy, desensitization or hyposensitization was first defined by Bostock(1819) which was later employed in the actual treatment by Noon (1911). IgE mediated allergic diseases are unique in that the sublethal parenteral administration of antigen, that is responsible for the disease, may render the patient specifically tolerant to that antigen as long as antigen is administered.

In one of the earliest controlled trials of immunotherapy (Brunn, 1949), 78% of the patients treated with house dust extract improved or remained free from symptoms compared with 22% of the control group, who did not receive immunotherapy.

Frankland and Augustin (1954) reported that 94% of their patients who received immunotherapy for allergic rhinitis and asthma had improvement in their symptoms.

Mc Allen (1961) found that house dust extract by injection was ineffective, while treatment by inhalation of an aerosol of house dust extract gave good though short lived results.

Smith (1971) demonstrated the role of immunotherapy in asthma, induced by house dust.

Aas (1971) found that 87% of 52 asthmatic children with house dust reactivity had a significant reduction in bronchial reactivity after treatment with house dust immunotherapy.

Though many studies have shown beneficial effects of immunotherapy some workers have reported the other way. Bruce et al (1977) treated a group of patients of allergic asthma (sensitive to ragweed) and there was no improvement in symptoms after immunotherapy. Causes of failure were attributed to improper detection of antigen, failure to include other antigen to which patients were sensitive or low dosage of antigen given.

A study to compare atopic patients who had received immunotherapy for 5 years with a group of atopic patients who had not received immunotherapy showed that there was no increase in the incidence of autoimmunity, collagen disorders, or lymphoproliferative

disorders in the treated group (Levinson et al, 1978).

Phanuphak and Kohler (1980) described 6 of 20 consecutive patients with polyarteritis nodosa in whome the onset of the vasculitis symptoms coincided with immunotherapy (within 6 months in 5/6) for presumptive atopic respiratory disease and vasculitis persisted despite discontinuation of immunotherapy. In 3/6 of the patients the respiratory symptoms (for which immunotherapy was given) were present for less than 5 months, suggesting that the symptoms were present or occurred during the prodrome of polyarteritis. So, they suggested not to initiate immunotherapy in any patient until at least one year after the onset of allergic respiratory illness.

While immunotherapy is carried out for all types of allergic disorders, immunotherapy is not recommended for treatment of food allergy. In case of food allergy, dietary exclusion of food is the treatment of choice.

MATERIAL AND METHODS

MATERIAL AND METHODS

The study was undertaken at the Allergy Clinic, Immunology and Biochemistry Laboratory, Department of Pediatrics, M.L.B. Medical College and Hospital, Jhansi.

SELECTION OF CASES

Present study comprised of patients having allergic disease - allergic asthma, allergic rhinitis and/or urticaria. Selection of cases was done from patients attending 'Allergy Clinic', Department of Pediatrics and also the referred cases from Pediatrics, Skin & V.D. and E.N.T. departments.

Diagnosis was based on detailed history, clinical examination and relevant investigations. Cases belonging to various socio-economic strata and occupations were included in the study.

Besides name, age, sex, address and socio-economic status of children, following facts were recorded in each case.

PRESENT, PAST AND FAMILY HISTORY

From parents or other family members detailed history was obtained regarding present illness, in chronological order. Emphasis was given to age of onset of first attack, frequency per year, symptom free period and precipitating factors for illness.

In the past history, history of worm infestation, atopy, asthma, bronchiolitis, and definitive history of pertussis and measles was elicited.

In the family history, history of atopy, urticaria, asthma, eczema, hay fever etc. was also recorded. An enquiry was made about the definitive history of tuberculosis of parents, siblings, near relatives and neighbourhood.

Relation of occurrence of symptoms with season, particularly months, hour of day, and place was outlined. Any recent change in residence and/or occupation was noted. History of any sort of animal contact was also enquired.

IMMUNIZATION HISTORY

History of immunization was taken from the parents or family members. For B.C.G. vaccination, confirmation was done from the scar mark.

SOCIAL HISTORY

A detailed account of the living conditions of the patient was made with special reference to the type of house (Kaccha/pacca), floor, water supply and toilet facilities. The status of hygiene was noted.

PHYSICAL EXAMINATION

Besides routine physical examination of the whole body, a record of the detailed examination of the system(s) involved was made. Thorough clinical examination

was done especially to observe skin changes, wheezing episodes, rhinorrhoea and sneezing.

Cardiovascular, respiratory and central nervous systems were examined in each case and abdomen was specifically examined for liver enlargement and spleen enlargement.

Children with bronchial asthma were assessed on the basis of their personal history of allergy, family history of allergy, degree of eosinophilia and the age of onset of illness.

Urticaria was considered acute if symptoms had been present for less than two months and chronic, if symptoms had persisted beyond two months before reporting. Physical test to determine the cause of urticaria included dermatographism test(by firm stroking of the skin) and challenge to cold, warm and pressure stimuli. The cold challenge was done by applying an ice cube to the skin for 2 to 5 minutes, as the skin rewarmed following removal of ice cube, presence of a wheal, if any, with its size, shape and surrounding erythema was noted. For warm stimulus, forearm was kept in warm water ($42^{o}C$) for ten minutes.

Foods were incriminated as the exciting cause of urticaria by history, ingestion test and elimination tests. The drug allergy was examined by oral challenge test in patient's with a history of urticaria, following ingestion

of certain drugs. Infection and worm infestation, as the inciting or perpetuating cause, were determined by history, physical examination and stool examination. Disappearance of urticaria on successful treatment of the infection or worm infestation confirmed the diagnosis.

WEIGHT

Weight was recorded nearest to 0.05 kg by using infant weighing scale, if the weight was less than 10 kg, while adult type weighing machine was used in children weighing more than 10 kg. In the later weight was recorded nearest to 0.1 kg.

INVESTIGATIONS

The following investigations were carried out.

1. Total and differential leucocyte count.
2. Haemoglobin.
3. Erythrocyte sedimentation rate (E.S.R.).
4. Absolute eosinophil count (AEC).
   AEC = TLC x percentage of eosinophil/100.
5. Chest skiagram.
6. Stool examination for ova and cysts.
7. Peak expiratory flow (litre/minutes) by peak flow meter.
8. Skin testing by modified prick method.

ALLERGENS

Commercially available prick test solutions containing aqueous allergen extracts, were used. The extracts contained 50% glycerol and were preserved in 0.4% phenol. Allergens included extracts from pollens, dust particles, insects, fungi and food substances. Details under each group are as follows :

Pollens : Hooptelia integrifolia, Pennisetum Typhides, Triticum Sativum, Parthenium hysterophorus.

Insects : Mosquito, Mite (D. Farinae).

Dust particles : House dust, Cotton dust, Hay dust, Paper dust.

Fungus : Aspergillus flavus.

Foods : Egg (whole), wheat, milk, rice, tomato, Banana, Lemon, Dal arhar, Dal urad, Masoor Dal.

Histamine solution served as positive control, while saline solution was used as negative control.

METHOD OF SKIN PRICK TEST (SPT), MODIFIED PRICK TEST

The most common site for skin testing is on the flexor side of the forearm. The skin was first cleaned with savlon and spirit, then the skin was marked, using a felt tip pen, 3-4 cm apart in two rows. Testing solutions (all the available allergen extracts) were subsequently placed at the sites marked with pen. The negative control (saline) was placed near the top of

the arm, followed by the allergen extracts, usually with the house dust, mite extract at the lower end, before the final positive control solution (Histamine). The test sites were approximately 4 cm apart, one small drop of test solution was applied on the skin with the tipe of plastic knob attached to the cap of bottle containing testing solution. To avoid difficulty in reading results, hairy skin was avoided.

A sterile lancet was introduced epicutaneously, through a drop of allergen extract, at an acute angle to the skin and shallow lift was made. The lancet was raised for a second before the skin was released. This was repeated for each drop of solution. Lancet was csrefully wiped off using cotton wool before using for each solution. Any excess solution remaining on the skin after the prick was made, was removed by placing a paper tissue over the upper arm for a moment or two.

The results were read after 20 minutes when positive reaction would appear as an induration surrounded by wheal and flare. Any wheal and flare produced by the negative control was substracted from any reactions produced by other allergens before they were assessed. The results were recorded by measuring wheal and flare with the help of divider and scale. Grading was done as follows :

Criteria	Symbols
Wheal and flare were only very small	+
Reaction larger but but not as large as the positive control.	++
Reaction similar to or greater than the positive control or reaction with pseudopodia.	+++

When a permanent record of the skin test reaction was required, the wheal and flare were closely encircled by a felt tip pen and a piece of clear adhesive tape was applied to the test site. Thus an image of the reaction was taken onto the tape which was then placed on patients record card.

Any contraindication to the test was not documented, except when there was a history of any anaphylactic reaction. In such cases histamine was injected with caution. Care was taken, all along that, that he blood was drawn during testing.

Patients were asked to discontinue (at least for 5 days) any medication that they were receiving for their allergic condition prior testing, as antihistamines and corticosteroids in high dosages could have affected the results of the test. Adrenaline injection and other resuscitative materials were kept available during testing.

PROCEDURE OF IMMUNOTHERAPY

Immunotherapy was performed to hyposensitize those patients who were shown to be sensitive to allergens as a result of skin prick testing.

Commercially available aqueous allergen extracts were used for specific hyposensitization.

The composition of each treatment set was determined from the reaction observed after skin prick test.

Initial treatment set consisted of 4 vials of 4.5 ml each.

Strength 1 : 1 + 24999 (0.004% w/v).

Strength 2 : 1 + 2499 (0.04% w/v).

Strength 3 : 1 + 249 (0.4% w/v).

Strength 4 : 1 + 49 (2% w/v).

Maintenance treatment set of one vial 4.5 ml containing extract of above mentioned strenth 4 was used. Bottle was shaken before use.

ADMINISTRATION AND DOSAGE

Aqueous allergen extracts were rapidly absorbed after injection.

The interval between the two successive injections was 2-3 days. It was never more than 7 days. If there was a gap of more than 7 days between two injections, for some reasons the dose given at the next injection was not increased.

Dosage scheme in children was as follows :

Vial/ Strength	Doses (ml)	Date	Individual dosage(ml) subcutaneous injections	Remarks
1	0.05			
1+24999	0.10			
Black label	0.15			
	0.20			
	0.25			
	0.30			
	0.35			
	0.40			
	0.45			
	0.50			
2	0.05			
1+2499	0.10			
Green label	0.15			
	0.20			
	0.25			
	0.30			
	0.35			
	0.40			
	0.45			
	0.50			
3	0.05			
1+249	0.10			
Blue label	0.15			
	0.20			
	0.25			
	0.30			
	0.35			
	0.40			
	0.45			
	0.50			

Continued...

4	0.05
1+49	0.10
Red label	0.15
	0.20
	0.25
	0.30
	0.35
	0.40

Maintenance therapy - Not given in any patient.

PRECAUTIONS

1. Injections were given in sterile condition. patients was advised not to perform heavy exercise after injection.
2. Injections were given at the extensor surface of upper arm, approximately between upper 2/3 and lower 1/3 of upper arm.
3. Injections were given subcutaneously by tuberculin syringe and patient was watched for 1/2 hour after injection.
4. Every course of hyposensitization treatment was initiated with the lowest dose (0.05 ml) from injection vial 1,2,3 and 4 subsequently.
5. Immunotherapy was not given in acute infections, severe acute asthma and when patient was on systemic steroids.

OBSERVATIONS

OBSERVATIONS

The present study was conducted in the Allergy Clinic of the Department of Pediatrics, and Immunology & Biochemistry laboratory of the Pediatrics Department, M.L.B. Medical College, Jhansi during the period of 14 months from October, 1991 to November, 1992 to analyse the spectrum of allergic disorders in patients attending the hospital and to evaluate the utility of allergens, by skin prick test, in various allergic disorders. Cases were selected from patients attending the allergy clinic and also few cases were referred from E.N.T. department.

Immunotherapy was done in two patients. In both the cases only the initial therapy was used and no maintenance therapy could be provided.

The present study comprised of total 57 cases of 2½ to 21 years age. Out of these, 48 cases were patients suffering from allergic disorders. Nine healthy cases of comparable age groups who had no present and/or past history of allergic disorders and had no history of allergy in the family, served as a control group in this study.

Out of 48 patients having allergic disorders, 43 patients were from 2½ to 15 years of age. Rest, 5 cases were between 16 to 21 years of age. Control group was having age ranging between 1 to 16 years.

A detailed history was taken and thorough clinical examination was done in each case. Microscopic examination of stool was performed to detect parasitic ova and cysts. Haemoglobin, total leucocyte count, differential leucocyte count and erythrocyte sedimentation rate were done and absolute eosinophil was calculated in all the cases. The skin testing was done by modified prick method with 21 allergens, to find out allergic factor(s).

TABLE I : Showing the distribution of patients according to age and sex.

Age groups	Number of cases		Total
	Male	Female	
0 - 3	1	-	1
4 - 6	9	4	13
7 - 9	8	4	12
10 - 12	12	3	15
13 - 15	2	-	2
16 - 18	3	-	3
19 - 21	1	1	2

1. AGE AND SEX DISTRIBUTION

Table I shows that out of 48 cases, 43 were under 15 years of age. According to sex, 36 cases were males and 12 were females, out of total 48 cases.

Youngest subject was 2½ years old while eldest was 21 years of old. Maximum cases were in their 4 to 12 years of life.

TABLE II : Showing general characteristics of cases.

Characteristics	No.of cases
Occupation :	
Students	45
Medicos	1
Miscellaneous	2
Residence:	
Pucca	45
Kaccha	3
Home set up :	
Rural	10
Urban	38
Dietary habits :	
Vegetarian	22
Non-vegetarian	23
Eggetarian	3
Methods of excreta disposal :	
Flush type	38
Field system	7
Daily system	3

2. GENERAL CHARACTERISTICS OF CASES

Table II shows that subjects belonged to various categories and had different general characteristics. Among 48 cases, 45 were students and one was a medical student. Majority of cases were having

their pucca houses. Only 3, out of 45 were living in kuccha houses.

TABLE III : Showing the age distribution of cases of different clinical groups.

Clinical group	Age in years								Total
	<1	2-3	4-6	7-9	10-12	13-15	16-18	>18	
Bronchial asthma	-	-	11	10	10	2	-	1	34
Allergic rhinitis	-	-	-	-	1	-	1	1	3
Urticaria	-	-	2	2	4	-	-	-	8
Allergic rhinitis + Bronchial asthma	-	1	-	-	-	-	2	-	3
Control	1	1	2	1	1	2	1	-	9
TOTAL	1	2	15	13	16	4	4	2	57

3. AGE IN DIFFERENT CLINICAL GROUPS

Table III shows the age distribution in different clinical groups. It shows that 31, out of 35 cases of bronchial asthma belonged to the age group of 4 to 12 years.

All the 8 cases of urticaria were above 3 years of age. Control cases were equally distributed in different age groups.

TABLE IV : Showing the age of onset of symptoms.

Clinical groups	Age of onset (years)							Total
	<1	2-3	4-6	7-9	10-12	13-15	16-18	
Bronchial asthma	3	10	7	9	3	2	-	34
Allergic rhinitis	-	-	-	1	1	-	1	3
Urticaria	1	1	1	2	3	-	-	8
Allergic rhinitis + Bronchial asthma	-	1	-	-	-	1	1	3
TOTAL	4	12	8	12	7	3	2	48

4. ONSET OF SYMPTOMS

Table IV shows the age at onset of symptoms in different clinical groups.

In asthma group, out of 35 cases, 29 had onset of wheezing or breathlessness before 10 years of age. Other clinical groups of allergic disorders did not show a definite pattern as far as the age of onset of symptoms was concerned.

TABLE V : Showing hemoglobin, blood counts, ESR in different clinical groups.

Clinical groups	No.of cases	Hb (gm%) Mean (Range)	TLC/cmm Mean (Range)	Differential leucocyte count(%) P Mean (Range)	L Mean (Range)	E Mean (Range)	M Mean (Range)	ESR (Wintrobe) mm in 1st hour Mean (Range)
Bronchial asthma	34	11.7 (8-16)	8417 (6100-11200)	52 (30-82)	39 (12-54)	8 (0-26)	1 (1-4)	28 (5-56)
Allergic rhinitis	3	13.0 (12-13)	7833 (6400-9500)	51 (48-62)	39 (26-52)	8 (3-10)	2 (1-3)	6 (2-10)
Urticaria	8	11.9 (9-14.4)	9025 (8200-10000)	57 (46-68)	38 (22-53)	5 (1-8)	-	24 (5-37)
Allergic rhinitis + Bronchial asthma	3	13.1 (12-14.8)	10067 (9300-10800)	68 (60-75)	24 (19-30)	8 (6-10)	-	25 (11-40)
Control group	9	10.1 (8-14)	8433 (7400-9400)	48 (28-60)	49 (38-70)	3 (1-8)	- (0-1)	40 (25-70)

5. HEMOGLOBIN LEVEL, BLOOD COUNTS AND ESR IN DIFFERENT CLINICAL GROUPS (TABLE V)

Cases of bronchial asthma showed higher mean eosinophil percentage (8%) count as compared to control group (3%). In urticaria eosinophil count was 5%, while allergic rhinitis showed 8% and allergic rhinitis associated with bronchial asthma had 8% eosinophilic count.

Hemoglobin percentage and leucocyte counts did not show much difference when different clinical groups were compared.

TABLE VI : Showing eosinophil count in different clinical groups.

Clinical groups	No.of cases	Mean (Range)	No.of cases having eosinophilia	
			7250/mm^3	7500/mm^3
Bronchial asthma	34	673 (0-2065)	28	19
Allergic rhinitis	3	595 (0-950)	2	2
Urticaria	8	451 (100-768)	5	5
Allergic rhinitis + Bronchial asthma	3	805 (606-1080)	3	3
Control	9	177 (0-360)	1	-
TOTAL	57		39	29

6. ABSOLUTE EOSINOPHIL COUNT IN DIFFERENT CLINICAL GROUPS

Table VI shows mean absolute eosinophil count in different clinical groups. Mean absolute eosinophil count was ⩽250/cmm in control group whereas it was more than 250/cmm in most of the allergic groups, being the highest in a group which had allergic rhinitis in conjunction bronchial asthma (805/cmm).

Twenty eight, out of 34 cases of bronchial asthma (82.3%); 2 out of 3 cases of allergic rhinitis (66.67%); 5 out of 8 cases of urticaria (62.5%) and all cases of allergic rhinitis with bronchial asthma (100%) showed eosinophilia with absolute count more than 250/cmm. Only 1 out of 9 control cases (11.11%) had eosinophilia.

Thus, 79.1% cases with allergic disorder (38 out of 48) showed eosinophilia as compared to only 11.1% (1 out of 9) control cases who had eosinophilia (eosinophil count ⩾250/cmm).

Nineteen cases of bronchial asthma (55.88%), 3 cases of allergic rhinitis (66.67%), 5 cases of urticaria (62.5%) and all cases of allergic rhinitis with bronchial asthma showed eosinophilia with absolute counts more than 500/cmm. None of the control cases showed eosinophilia of more than 500/cmm.

TABLE VII : Showing skin prick test in various clinical groups with and without intestinal parasitic infections.

Clinical groups	Intestinal infection		A.E.C./ cmm	No.of cases of +ve SPT
Bronchial asthma(N=17)	None	-13*	668	7
	Protozoal	- 2	432	1
	Helminthic	- 2	936	1
Allergic rhinitis(N=1)	None		684	1
Urticaria (N=5)	None	- 4	605	None**
	Protozoal	- 1	100	1
Allergic rhinitis + Bronchial asthma	None	- 1	1080	1

* SPT done only in 11 cases out of 13.
** SPT was not done.

7. ABSOLUTE EOSINOPHIL COUNT IN DIFFERENT CLINICAL GROUPS AND ITS RELATION TO INTESTINAL PARASITIC INFECTION/INFESTATION AND TO SKIN PRICK TEST (TABLE VII)

Out of 34 cases of bronchial asthma, stool test for ova and cysts by light microscopy was done in 17 cases. Out of 17 cases for whom stool test was done, only 4 cases (23.5%) were positive, 2(11.76%) cases for cyst of giardia lambia and 2(11.76%) cases for helminthes (one for ova of ancylostemata duodenal and one for Ascaries lumbricoides).

Those cases of bronchial asthma whose stool test was positive for ova/cyst of helminthes, showed higher absolute eosinophil count than those with negative

stool test. Moreover, stool positive for protozoal ova/cyst did not show this difference.

Skin prick test positivity was not influenced by stool parasitemia, as skin prick test positivity was 63.6% in stool negative cases and 50% each in protozoal and helminthic infestations.

TABLE VIII : Reaction of histamine in various allergic disorder at different age groups.

Age groups (years)	Bronchial asthma (mm)	Allergic rhinitis (mm)	Urti-caria (mm)	Allergic rhinitis + bronchial asthma (mm)	Lactose intolerance with milk allergy (mm)
0-1	-	-	-	-	1
2-3	-	15	-	15	-
4-6	12.6(6-18)	-	-	-	-
7-9	13.2(5-30)	-	-	-	-
10-12	9.3(3-15)	8	8	-	-
13-15	7.0(6-8)	-	-	-	-
16-18	10	6	-	8(6-10)	-
18-21	20	6	-	-	-

8. HISTAMINE REACTIVITY IN DIFFERENT CLINICAL GROUPS AT DIFFERENT AGES

Table VIII shows histamine hyporeactivity in infancy but after 2 years of age histamine reactivity approximates to adolescence.

Maximum reactivity to histamine was noted in 7-9 years age group (30 mm) and average was 13.2 mm with a

range of 5-30 mm. The least reactivity was noted in infancy.

TABLE IX : The study group and family history of allergy.

Clinical groups	No.of cases	Sex		No.of cases with +ve F. history of allergy.
		Male	Female	
Bronchial asthma	34	25	9	12
Allergic rhinitis	3	2	1	2
Urticaria	8	5	3	1
Bronchial asthma + allergic rhinitis	3	3	-	1
Control	9	-	-	-
TOTAL	57	35	13	16

9. RELATION OF FAMILY HISTORY OF ALLERGIC DISORDER(S) WITH PATIENTS SYMPTOMATOLOGY

As shown in table IX, allergic cases were divided in different clinical groups viz., bronchial asthma, allergic rhinitis, urticaria and allergic rhinitis with bronchial asthma.

The family history of allergic disorders was positive in 12 out 34 cases (35.24%) of bronchial asthma, 2 out of the 3 cases of allergic rhinitis (66.67%) cases, and one out of 3 cases of allergic rhinitis associated bronchial asthma (33.33%) and One (12.5%) out of 8 cases of chronic urticaria.

TABLE X : Showing SPT positivity in different clinical groups.

Clinical groups	Total No.of cases	No.of cases in which SPT done	SPT positive cases
Bronchial asthma	34	25	15
Allergic rhinitis	3	3	3
Urticaria	8	2	1
Allergic rhinitis + bronchial asthma	3	3	2

10. SKIN PRICK TEST POSITIVITY IN RELATION WITH DIFFERENT CLINICAL GROUPS(TABLE X)

Out of a total of 48 cases of allergic disorders, skin prick test was performed in only 33 cases, out of which 21 (63.6%) cases were positive for skin prick test.

All the 3 cases of allergic rhinitis were positive for skin prick test, 2 out of 3 cases of 'allergic rhinitis with bronchial asthma' were positive for SPT and 15 out of 25 cases of bronchial asthma were positive for SPT.

TABLE XI : Indicating the distribution of SPT positive cases in respiratory allergy.

Allergy groups	No.of cases		
	Male	Female	Total
Dust	4	-	4
Pollens	1	-	1
Insects	-	1	1
Fungus	-	-	-
Food(s)	-	-	-
More than one group	8	3	11

11. DISTRIBUTION OF SKIN PRICK TEST POSITIVE CASES IN RESPIRATORY ALLERGY GROUP(ASTHMATICS WITH OR WITHOUT NASAL ALLERGY)

Depending upon the skin sensitivity test results, patients were divided as follows : dust group, (sensitivity to house dust, hay dust or cotton dust), pollen group, food group; insect group (cases positive for more than one group were assigned a separate group.

Table XI shows that most (64.7%) cases were positive for more than one group of allergens, followed by dust group which showed SPT positivity in 23.5% cases of respiratory allergy.

TABLE XII : Showing SPT positive cases in allergic rhinitis and urticaria.

Allergen groups	No.of cases		
	Male	Female	Total
URTICARIA			
Food	-	1	1
ALLERGIC RHINITIS			
Pollens	-	1	1
More than one group	1	1	2

12. DISTRIBUTION OF SKIN PRICK TEST POSITIVE CASES IN ALLERGIC RHINITIS AND URTICARIA

Table XII shows that 1 case of urticaria was positive for food (Dal Urad). Similarly one case of allergic rhinitis was positive for pollens and remaining 2 cases were positive for more than one group of allergens.

TABLE XIII : Showing the number of cases sensitive to more than one component of dust.

Component	Number of cases		
	Male	Female	Total
Paper + House dust	1	-	1
Paper + Hay dust	1	-	1
Paper + Hay + House dust	-	1	1
TOTAL	2	1	3

13. CASES SENSITIVE TO MORE THAN ONE COMPONENT OF DUST

Table XIII shows that paper dust had contribution in all the 3 cases sensitive to dust components. Contribution of hay dust and house dust in causing allergy was seen in 2 out of 3 cases.

TABLE XIV : Showing the number of cases sensitive to more than one component including one component of dust in asthmatic disease and allergic rhinitis cases.

Dust with other components	Number of cases		
	Male	Female	Total
Dust + Pollen	2	2	4
Dust + fungus	1	-	1
Dust + insect	3	-	3
Dust + Insect + Pollen + Fungus	1	-	1
TOTAL	7	2	9

14. SENSITIVITY TO MORE THAN ONE COMPONENT OF ALLERGEN.

Table XIV shows that 9 cases were positive for SPT and all had sensitivity to one dust component plus one other component viz. pollen, insect and fungus or a combination of more than two components.

TABLE XV : Showing prick test response to various allergens in bronchial allergy.

Allergens	Positive for SPT	
	No.	Percentage
Insects	8	28.57
Dusts	14	50.00
Foods	-	-
Pollens	6	21.43
Fungi	2	7.14
Dusts + Insects	16	57.14
Dust + Pollens	16	57.14
Dusts+Insects+Pollens	17	60.71
Dusts+Insects+Pollens+Fungi	17	60.71

15. PRICK TEST RESPONSE TO VARIOUS ALLERGENS

SPT was done on 28 cases between the ages of 3 and 21 years of respiratory allergy(Asthmatics with or without rhinitis) (Table XV).

Table XV shows the SPT response to various groups of allergens. It was seen that dust and insect allergens were the most common allergens while food was not responsible for any case. Fifty percent gave a positive reaction to dust, 28.57% to insect allergens, 25.43% to pollens, 7.14 to fungi and there was no case to have positive reaction to food. Reactions to insect allergens together with dust allergeus were positive in 57.14% cases. Pollens together with dust allergens were

positive in 57.14% cases on skin prick testing.

TABLE XVI : Prick test response to dust allergens in bronchial/respiratory allergy.

Dust	Positive SPT	
	No.	Percentage
House dust	11	39.28
Paper dust	6	21.43
Cotton dust	5	17.86
Hay dust	5	17.86

16. PRICK TEST RESPONSE TO DUSTS IN RESPIRATORY ALLERGY.

Table XVI shows that the majority of the cases were sensitive to house dust followed by these sensitive to cotton dust.

TABLE XVII : Showing patients sensitive to single allergen.

Sl. No.	Age/Sex	Clinical diagnosis	SPT positive for
1.	12/M	Bronchial asthma	House dust
2.	14/M	Bronchial asthma	House dust
3.	12/F	Allergic rhinitis	Triticum sativum
4.	4½/M	Bronchial asthma	House dust
5.	8/F	Bronchial asthma	D. Farinae

17. SENSITIVITY TO SINGLE ALLERGEN (TABLE XVII)

Sensitivity to a single allergen was rather infrequent. Table XVII shows that 5(23.8%) cases were positive to single allergen out of 21 cases. Among these positive for single allergen, house dust sensitivity was the commonest (3 out of 5 cases).

TABLE XVIII : Showing relation of precipitation factor, seasonal variation, family history and SPT in bronchial asthma.

Sl. No.	Age/ Sex	Precipitating/ causative factors	Seasonal variation	Family history for allergy	Skin Prick test
1.	8/F	Cold, curd, ice cream, house dust	More in spring season	+ve	+ve
2.	12/M	Ice cream, cold	More in winter	+ve	+ve
3.	10/M	Cold, dust, exercise	-do-	+ve	+ve
4.	4/M	Banana, cold, ice cream	-do-	-ve	+ve
5.	10/M	Banana, orange, mango toffee, egg, dust, cold, exercise.	No	-ve	-ve
6.	10/M	Cold, ice cream	No	+ve	-ve
7.	7/M	Cold	More in winter	+ve	+ve
8.	7/M	NO	-do-	-ve	-ve
9.	12/M	Cold, pea, tomato	-do-	-ve	+ve
10.	8/F	Cold	-do-	+ve	+ve
11.	6/F	No	-do-	-ve	+ve
12.	7/F	Cold, fever	-do-	-ve	-ve

contd ...

13.	9/M	Cold	More in winter & rainy season	-ve	-ve
14.	10/M	Milk,gur,oil	More in winter	-ve	-ve
15.	8/M	Ice cream,cold,rice	-do-	-ve	-ve
16.	20/F	Coryza	More in Spring & changing season	-ve	-ve
17.	6/M	Rice,muli,cold	More in winter	-ve	-ve
18.	4½/M	No	No	-ve	+ve
19.	7/M	Cold	Rains	+ve	+ve
20.	14/M	Cold	More in summer	-ve	+ve
21.	5/M	Cold	More in winter	-ve	-
22.	12/M	NO	No	-ve	+ve
23.	8/M	Cold	No	-ve	-
24.	12/M	Cold,viral infection	More in winter	-ve	+ve
25.	5/M	Cold	-do-	+ve	-
26.	5/M	Ice, cold	No	+ve	-
27.	4/M	Cold,ice cream	More in winter	-ve	-
28.	5/F	No	No	-ve	-
29.	7/F	Rice, cold	More in winter	+ve	-
30.	4/M	Ice cream,cold	-do-	-ve	-
31.	6/F	No	No	+ve	-ve
32.	13/M	Curd,house dust	More in winter & rainy season	+ve	+ve
33.	11/M	Ice,cold,dust	More in winter	+ve	+ve
34.	10/M	No	No	-ve	-

18. RELATION TO PRECIPITATING FACTORS, FAMILY HISTORY OF ALLERGY, SEASONAL VARIATION AND SPT IN BRONCHIAL ASTHMA

Table XVIII shows that precipitating factors were present in majority of cases i.e. 28(82.35%) out of 34 cases of bronchial asthma. In majority (85.7%) of patients cold was the precipitating factor, second in importance was ice-cream (32.4%) and last was the viral fever in 10.7% cases.

Seasonal variation was present in 76.47% cases and in a great majority (80.7%) of them frequency of episodes increased during winter season. In rainy season 11% patients showed increased symptomatology. In spring season 7% and in summer 3.8% patients suffered from increased frequency of symptoms.

Family history of allergic disorder was positive in 13 out of 34 cases of bronchial asthma (38.2%).

SPT was done in 25 cases of bronchial asthma and was positive in 15(60%) cases. On evaluating skin prick test positivity in positive family history cases, SPT positivity was seen in 70% (7/10) cases as compared to 60% positivity seen when positive family history was not present.

TABLE XIX : Showing features of urticaria cases.

Clinical entity	Precipitating or causative factors	Family history of allergy	SPT	Cryostimulation test	Heat emmersion test
Chronic urticaria with eosinophilic edema	Ice, brinjal Khatai	+ve	+ve	-	-
Chronic urticaria	No	-ve	-ve	-ve	-ve
Chronic giant urticaria	No	-ve	-	-ve	-ve
Giant urticaria with angioneurotic edema	No	-ve	-	-ve	-ve
Giant urticaria	No	-ve	-	-ve	-ve
Cold urticaria (chronic)	Bath, banana, chocolate	-ve	-	+ve	-ve
Dermatographism	Trauma, Prick	-ve	-	+ve	-ve
Chronic urticaria	No	-ve	-	+ve	-ve

19. FEATURES OF URTICARIA PATIENTS

All patients included in the study were of recurrent chronic urticaria. Table XIX shows that precipitating factors were present in 3 cases only.

Family history of allergy in cases of urticaria was present only in one case.

In 7 cases, cryo stimulation was done and two cases showed urticarial lesion after applying ice or cooled water on parts of the body.

D I S C U S S I O N

DISCUSSION

A prospective study was carried out to assess skin prick test in various allergic disorders, role of age on skin prick test reactions, hyposensitization in allergic diseases, absolute eosinophils counts and role of intestinal parasites in allergic diseases.

A sample of 57 children which included 48 allergic cases and 9 children for control were considered for the study. All the cases were below 21 years of age.

The study group was divided into following categories of patients. Bronchial asthma (34), allergic rhinitis (3), urticaria (8) and allergic rhinitis with bronchial asthma (3).

The study was undertaken at the Allergy clinic, Immunology and Biochemistry laboratory, Department of Pediatrics, M.L.B. Medical College and Hospital, Jhansi.

Diagnosis was based on detailed history, clinical examination and relevant investigations. From parents or other attendants detailed history was obtained regarding present illness in chronological order. Emphasis was given to age of onset of first attack, frequency per year, any precipitating factor, history of worm infestation, family history of allergy and family history of tuberculosis.

Hemoglobin, blood leucocyte count, differential leucocyte count and erythrocyte sedimentation rate were done. Absolute eosinophil count was calculated in all the cases. An attempt was made to show the relationship of eosinophils with allergic disorder and intestinal parasitic infection.

Stool test was done by light microscopy for ova and cyst in a few cases.

Allergy testing was done by modified prick test method using prick test solutions, which were glycerinated aqueous allergen extracts. Prior to subjecting patients to skin prick test, they were asked to stop antihistamines and steroid atleast five days before tests, which they were taking. The results were measured by divider and scale and both wheal and flare were included in measurement. Prick test was done only in the study group. Saline was used for negative control and histamine for positive control.

Immunotherapy was done by commercially available aqueous allergen extracts, to hyposensitize, those patients who were diagnosed as being sensitive to allergen, following skin prick testing.

On the basis of observations depicted in table I to XX, various interferences have been drawn and discussed under different headings.

FAMILY HISTORY OF ALLERGY AND SEX

As shown in table IX, none of the control cases

had positive family history of allergy. In present study positive family history of atopy was found in 35.3%, 66.7%, 12.5% and 33.3% cases of bronchial asthma, allergic rhinitis, urticaria and bronchial asthma in conjunction with allergic rhinitis, respectively.

Cooke and Vander Veer (1916) showed positive family history of atopy in 48% cases of bronchial asthma, as compared to 7% in control cases. In 1920, again 48% cases of atopy was reported by Adkinson in a study of 400 asthmatic patients. In 1924, Spain and Cooke observed 58.4% cases with family history of atopy in patients of hay fever and asthma. But, in the present study family history of atopy was positive in 35.3% cases of bronchial asthma and in 66.7% cases of allergic rhinitis. Bruce (1958) observed that nearly 50% patients of asthma had family history of allergy. Lubs (1971) observed that concordance of asthma in monozygotic twins was 25%, but it was 16% in the dizygotic twins. They suggested that genetic factors would be less important, and environmental factors more important. They also evaluated that the risk of atopy (hay fever, asthma or eczema) was about 33% when both parents were atopic, and 24-29% when one parent was atopic, and where neither parent was atopic, the risk was in between 14-25%. In contrast, present study noted a family in which one parent (father) was atopic and his two children (one male and one female) were atopic, out of a total of 3 children he had.

TABLE XXI : Family history of allergy.

Type of allergy	Percentage of positivity				
	Present study	Jha et al(1975) Varanasi	Kasliwal et al (1959) Jaipur	Chaubey et al (1973)	Vishwa-nathan (1964)
Asthma	35.3	61.0	40.0	41.0	38.0
Rhinitis	66.7	45.0	-	-	-
Asthma with rhinitis	33.3	76.5	-	-	-
Urticaria	12.5	-	-	-	-

Rackeman et al (1952) from a large survey concluded that atopy was associated with 82% asthmatic children. On the other hand Pife et al (1981) could not detect atopy in more than 20% of asthmatic children studied. Arsdel et al (1959) observed that offsprings of a single atopic parent had about 50% chances of being atopic and if both parents had atopy, the chances were even greater.

Koning and Godfrey (1974); Bazarel et al (1974) observed that there was a tendency for offspring to show the same symptoms and to react to same antigens as their parents did.

In the present study it was observed that naso-bronchial allergy was common in male as compared to female (3 : 1) children. Similar observations have been made by Vishwanathan (1964), Wig and Guleria (1964) and Agnihotri and Singh (1971).

Urticaria was considered acute if symptoms were present for less than 2 months. Urticaria considered

chronic if symptoms persisted beyond 2 months and recurrent if the recurring episodes of urticaria were of shorter duration than symptom free periods between episodes (Kauppinen et al, 1984). Table XVII shows that all the 8 cases in present study had recurrent urticaria. The etiology could possibly be established in only 3(37.5%) cases. One was skin prick test positive and the other two cases showed symptoms on cryostimulation. Champion et al (1969) could not find the cause of urticaria in 75% of their cases. But Pasricha (1980) and Kauppinen (1984) were able to define an etiology in about half of the cases of urticaria. In the present study etiology of urticaria was observed in 37.5% cases. Champion et al (1969) also stated that origin of acute urticaria was usually detectable.

Helpern (1965) observed that at least half of all children presenting with urticaria were atopic. Champion et al (1969) observed that there was no solid evidence that patients with history of atopy had an increased incidence of chronic urticaria. Present study also favours Champion's observation, since there were only 12.5% cases of urticaria who had a family history of atopy (Table IX).

Urticaria was some what more frequent in females than in males (Sheffer, 1975), but the present study male dominance (5 males out of 8 cases) as shown in table IX.

BLOOD COUNTS IN DIFFERENT CLINICAL GROUPS

As shown in table V, none of the patients or control cases had hemoglobin below 8 gm%. All the patients and control cases had a total leucocyte count within the normal range. However, maximum mean eosinophil count of 8% was seen in bronchial asthma, allergic rhinitis and allergic rhinitis associated with bronchial asthma. Acharya (1983) also reported eosinophilia of 10-14% in peripheral smear in his study on childhood asthma.

Increased absolute eosinophil count has for long been shown to be the common accompaniment of atopy. Table VI shows absolute eosinophil count of more than 250/cmm in 79.2% allergic cases in the present study.

Approximately 82.3% cases of bronchial asthma were having absolute eosinophil count more than 250/cmm; cases of allergic rhinitis had 66.6% and those of urticaria had 62.5%. Allergic rhinitis with bronchial asthma cases showed eosinophilia in 100% cases. In control group eosinophilia was in 11.1% cases. Eosinophilia was of the highest degree in those cases of bronchial asthma who had associated rhinitis (mean eosinophil count 805/cmm as shown in table VI). Association of eosinophilia with allergic disorder was also reported by Bray (1931) and Smith (1931). Lowell (1967) and Sharma (1974) in their study reported eosinophilia in majority of the cases of bronchial asthma. In the present study also, there were 28, out of 34 cases of bronchial asthma who had eosinophilia of more than 250 cells/cmm.

In the present study 82.3% patients suffering from bronchial asthma had eosinophilia (AEC 7250/cmm) while in previous studies on asthma patients, Sharma (1974), Arshad et al (1981) and Acharya and Saxena (1983) reported eosinophilia in 71.8%, 56% and 45.3% cases of asthma respectively. Some authors are of the opinion that eosinophil count may be lacking as a result of intercurrent infections (Lowell, 1967). However eosinophilia may be seen intermittently. Normal results should be obtained 2-3 times before it is concluded that eosinophilia is not present (Nelson, 1983).

Luksza and Jones (1982) observed eosinopenia (AEC 170/cmm) in acute cases of asthma and eosinophilia in chronic and stable cases of asthma. Authors observed that 14 out of 16 cases of acute and savere asthma had absolute eosinopenia (two cases had whole blood eosinophil counts of only 170/cmm). In the present study a few cases of asthma (8.8%) had absolute eosinopenia.

Eosinophilia was a characteristic finding in patients with atopic disorders, including dermatitis, rhinitis and asthma (Stickney et al, 1944). Similarly present study showed eosinophilia (7250/cmm) in 66.7% cases of allergic rhinitis, 82.3% cases of bronchial asthma, 62.5% cases of urticaria and 100% cases of allergic rhinitis associated with bronchial asthma. Tandon and Saha (1987) observed that 55% cases of bronchial asthma had high

(7500/cmm) eosinophil count. The present study showed 19 cases out of 34 cases (58.88%) with such high eosinophil counts.

INTESTINAL PARASITIC INFECTION IN ALLERGIC DISORDERS

Intestinal parasitic infection was diagnosed with the help of microscopic examination of stool. But of 48 cases, stool examination was done only in 28 cases and only 5 cases (17.8%) cases were positive for ova/cysts.

In bronchial asthma 4 out of 17 cases (23.5%) were positive on stool examination (two for cysts of Giardia lambia, one for Ankylostoma duodenale and one for cyst of Ascaris lumbricoides). Absolute eosinophil count was higher in cases having helminthic infestation than in those who had protozoal infection or those who had stool negative for ova and cyst (Table VII). However, there was higher absolute eosinophil count in stool negative cases than in those cases whose stool was positive for cysts of giardia lambia.

In allergic rhinitis stool examination was done in one case and that was negative for ova/cysts.

Stool examination was done in 5 out of 8 cases of chronic urticaria. Only 1(20%) case was positive for cysts of giardia lambia. Even in this case, after the treatment for giardia lambia, recurrence of urticaria did not stop.

There was no improvement in the symptoms even after successful treatment of parasitic infection.

Pasricha (1972) concluded that intestinal infection

did not constitute a significant cause of urticaria. This was also noted in the present study, since the symptoms of urticaria did not subside even after a successful treatment of intestinal parasitic infection. In contrast some workers have reported intestinal parasites, particularly ascaris, as an important cause of urticaria (Gabr, 1976). Pasricha (1979) in yet another study found only 1.4% cases in whom elimination of parasites had resulted in relief of urticaria.

Veronesi (1982) and Hamriek (1987) reported a relationship existing between urticaria and giardiasis. In present study 20% cases of chronic urticaria were positive for cysts of Giardia lambia, on stool examination by light microscopy. But, in these cases peripheral eosinophilia (AEC 100/cmm) was lower than that found in stool negative cases (AEC 605/cmm). Twarog (1983) suggested to consider the presence of parasitic infection in individual cases of urticaria who came from an endemic area, had peripheral eosinophilia and had elevated IgE levels. In contrast, present study showed eosinopenia in giardia infected cases as compared to stool negative cases. Block (1985) reviewed various studies and found that data illustrated discordance between parasitism and the presence of allergic diseases. This finding was confirmed from the present study.

RELATION OF INTESTINAL PARASITEMIA WITH SKIN PRICK TEST

Skin prick test by modified method was done in 33 allergic cases and an attempt was made to correlate the skin prick test positivity with intestinal parasitic infection (Table VII).

In bronchial asthma, stool examination was done in 17 cases and skin prick testing was done in 15 cases. Out of 17 cases, 13(76.5%) cases were negative on stool examination and skin testing was done in 11 cases. Out of 11 cases 7 were positive on skin prick test (63.6%). In two (11.7%) cases stool test was positive for giardia lambia and in both cases skin testing was done : only one case had positive reaction.

Two cases (11.7%) were positive for helminthic infestation i.e. one for Anycylostoma duodenale and other for ascaris lumbricoides. In both the cases skin prick test was done and one case showed positivity.

Stool examination was done in one case of allergic rhinitis and it was negative for ova/cyst but on skin prick testing the child showed positive result(100%). Similarly, in 5 urticaria cases stool examination was done, one case (20%) was positive for giardia lambia and rest (4(80%) cases were negative. Skin testing was done only in stool positive cases and that showed positive reaction. But, in a case of bronchial asthma associated with allergic rhinitis even though stool examination was negative yet the skin prick test was positive. Thus it

seems that intestinal parasitic infection/infestation has no relation with skin prick test positivity or negativity.

SKIN PRICK TEST REACTION

In the present study, skin testing was done by modified prick method on volar surface of forearm. Pherwani (1985) in his study of asthmatic children in Bombay followed the skin prick method described by Shivpuri (1965). Ajay Shanker et al (1979), Agarwal et al (1974), Jha et al (1975) however, performed the intradermal test for allergy testing.

The inclusion of histamine in skin prick testing was recommended for optimal evaluation of allergen hypersensitivity (Nelson, 1983 and Malling, 1984). Some investigators advocated semiquantitative grading of skin test reactions to allergens based on the presence and size of a positive control reaction (Aas, 1980). In the present study histamine was used as a positive control. Casale et al (1984) proposed to use codein as a positive control which triggers mast cells via specific cellular receptors while histamine is an end organ mediator.

In the western countries erythema forms the major parameter for positivity (Norman, 1980 and King et al, 1962) but Agrawal (1982) suggested to measure wheal in Indians, as erythema on skin was not always intense. In the present study both flare and wheal were measured.

It has been demonstrated that histamine reactivity was lower in infants and elderly. Brociek (1985) has proposed that reactivity to histamine increased until adulthood was reached and then decreased after 50 years, till a plateau was reached at about 60 years of age. Since the size of skin prick test reaction to histamine varied with age, the interpretation of skin prick tests should not only take into account the wheal size but rather a ratio between histamine induced and allergen induced wheals" was the opinion of Lessof et al (1980).

In the present study out of 15 patients of asthma who showed reactivity against one or more allergen, 12 (63.6%) were resident of urban area while one belonged to rural locality (33.3%). Residency in urban areas was an indicator for increased reactivity as proved by multivariate analysis (Gergen, 1986). Linna (1974) also found in his study that skin reactivity was more in urban dwellers. Thus, the present study showed similarity with the studies of Gergen and Linna. Smith et al (1982) have reported same or a lower incidence of reactivity in the rural population.

In the present study all skin prick sensitivity tests were conducted in between 10 AM and 1 PM. It has often been questioned whether circadian rhythm affects skin prick test results. Reinberg et al (1985) demonstrated maximum skin reactivity between 7-11 PM and minimum at 7 AM.

Lee et al (1977) confirmed these findings and suggested that false negative readings could be the result of early morning office hours. In contrast, Vichyanand et al (1989) showed that there was no significant morning-evening variation in skin prick test results.

Majority of nasobronchial allergy cases had only bronchial asthma (85%). A smaller group had rhinitis alone (7.5%) and a similar number had rhinitis combined with asthma (7.5%). In the latter group, rhinitis either preceded or accompanied the development of asthma (Table IX). But Jha et al (1975) found in their study - 17.9% cases of rhinitis, 36.5% asthma and 45.5% asthma associated with rhinitis. Wig and Guleria (1964), Agnihotri and Singh (1971) reported similar results as reported by Jha et al (1975). Present study included only children whereas other studies included both children and adults. That may explain more cases of bronchial asthma in the present study. Age of onset of symptoms of rhinitis was towards the higher side in contrast to cases of bronchial asthma (Table IV).

REACTION OF HISTAMINE AT DIFFERENT AGES

Skin prick test was done in a 6 months old male child, clinically suspected to be suffering from milk allergy. The child showed a pin point erythema at the prick sites of histamine, milk and saline. The reaction occurred after 20 minutes and measured about 1 mm (Table VIII). This confirmed the observations that skin test

reactivity to histamine was lower in infants by comparison with adults (Menardo et al, 1985 and Barbee et al,1976).

Asperen et al (1984) gave the details of end organ responsiveness in infants and elderly individuals to mediators of inflammation.

In asthma cases reactivity to histamine was somewhat increased even upto the age of 21 years, but in rhinitis and rhinitis with asthma cases reactivity decreased with age, maximum being at the age of 3 years (Table VIII). In asthma cases, maximum reactivity was 30 mm in 8 years old (both flare and wheal) with multiple pseudopodia. Brociek et al (1985) observed increased histamine reactivity from infancy to adulthood. Similar was the observation of Barbee et al (1981). Present study also showed the same results as far as asthma patients were concerned.

SKIN PRICK TEST IN RESPIRATORY ALLERGY

In the present study, there were 34 cases of asthma and prick test was done in 25 cases. Out of 25 cases 15(60%) were positive (Table X). Sethi et al (1986) reported positive allergy test in 41.27% cases of asthma. Shivpuri and Singh (1965) reported positive response in 32.5% cases of bronchial asthma(children & adult). Ajay Shanker et al (1979) found somewhat low (15.07%) positive results in asthmatics(children and adults). In the present study higher positive results

could be seen because the sample was selected from age group of 4 years to 21 years, while other authors studies involved mainly the adult cases.

Bronchial allergy cases, mostly, gave positive reaction to more than group of allergen (64.7%) and single allergen positive reaction was present in 23.5% cases only, of which 75% were because of house dust. So, it was concluded that bronchial allergy occurred usually due to multiple allergens (Table VI).

In the present study 14 cases (50%) of bronchial/ respiratory allergy were found to be reacting positively to dust allergens. The next common allergen was an insect extract (28.57%) (Table XV).

Pherwani et al (1985) found 81.25% cases allergic to insects. The next common allergen was dust (71.9%) in his study.

In the present study reaction to dust and insect allergens, taken together, was positive in 57.14% cases of respiratory allergy. This figure was slightly lower than that of Jha et al (1975) who found an incidence of 73.1%. But, Pherwani et al (1985) showed 81.25% cases allergic to insects and dust allergens, taken together.

Higher incidence of dust allergy in the present study could be explained on the basis of dry, dusty weather of Jhansi and a similar situation was described by Jha et al (1975) from Varanasi.

TABLE XXII : Prick test response to various allergen in bronchial allergy.

Allergen	Positive Prick test(Percentage)		
	Present study	Pherwani et al (1975) Bombay	Jha et al (1975) Varanasi
Dust	50.00	71.90	73.10
Insects	28.57	81.25	-
Foods	-	62.50	-
Pollens	21.43	43.75	-
Fungi	7.14	40.60	-
Dusts & insects	57.14	81.25	73.10
Dusts & Pollens	57.14	75.00	-
Dust + Insects + Pollens	60.71	90.60	-
Dust + Pollen + Insects + Fungi	60.71	93.75	-

Fourteen (50%) cases gave positive reaction to dust. Reaction to house dust was most common and was seen in 39.28% cases in the present study. The next common dust allergen was paper dust seen in 21.43% cases, followed by cotton dust in 17.86% cases and hay dust in 17.86% cases. In the study of Jha et al (1975) reaction of house dust was more common, similar to that seen in the present study.

Two cases (7.14%) gave positive reaction to fungus (aspergillus flavus). In Shanker's study (1979) antigens derived from cladosporium, Alternaria, Aspergillus flavus and Aspergillus fumigatus were common among twelve

fungus allergens used for skin testing. But in the present study only one fungus allergen i.e. Aspergillus flavus was used.

TABLE XXIII : Skin prick test response to pollens, insects, food and fungi in comparison with various authors.

Allergens	Present study (SPT %)	Pherwani et al (1985) Bombay(SPT%)	Interadermal test %
POLLENS			Shanker
Holptelea integrifolia	3.57	-	-
Pennisetum typhoides	7.14	12.50	1.40
Triticum sativum	7.14	-	-
Parthenium-Hysterophorous	17.85	6.25	2.10
INSECTS			
Mosquito	10.71	37.50	12.50
Mite(D. Farinae)	14.28	31.30	-
Dusts			Jha
House dust	39.28	31.25	34.80
Cotton dust	17.86	37.50	19.60
Hay dust	17.86	-	-
Paper dust	21.43	18.80	18.80
FUNGUS			Agrawal
Aspergillus flavus	7.14	-	12.00
FOODS			
Egg (whole)	-	-	-
Wheat	-	-	-
Milk	-	-	-
Rice	-	-	-
Tomato	-	-	-
Banana	-	-	-
Lemon	-	-	-
Dal arhar	-	-	-
Dal Urad	-	-	-
Masoor Dal	-	-	-

TABLE XXIV : Comparison of results of dusts allergy reported by various investigators on bronchial allergy.

Dust	Present study	Sethi et al(1975) Raipur	Shanker (1979) Gwalior	Mittal (1979) Kanpur	Jha (1975) Varanasi	Shivpuri (1975) Delhi	Shivpuri (1971) Delhi	Pherwani (1985) Bombay
House dust	39.28	42.00	3.50	11.10	34.80	32.60	34.60	31.25
Paper dust	21.43	52.00	-	-	18.80	-	-	18.80
Cotton dust	17.86	10.00	4.20	-	19.60	9.50	20.20	37.50
Hay dust	17.86	-	-	-	-	-	-	-

INSECTS IN RESPIRATORY ALLERGY

Eight cases (28.5%) gave positive reaction to insects antigen. Positive reaction to mite (D. Farinae) was the commonest among insect allergens (14.28%) in the present study. Skin sensitivity to cockroach allergen followed by mosquito, house flies and D. farinae allergens, in that order, was seen in the study of Pherwani et al (1985). In Shanker's study (1979) the corresponding order was : house flies, mosquitoes and cockroach antigens. Present study could not be well compared to other series, since only two insect allergens viz. mite and mosquito were used.

FOOD AND ALLERGENS

In the present study, positive reaction to food allergens in bronchial allergy cases was not observed and this was in absolute contrast to previous studies. Pherwani et al (1985) reported 62.5% cases with positive reaction to one or more food allergens in bronchial allergy cases. Pherwani et al (1985) observed 90% of children below 6 years giving positive reaction to food allergen. In contrast, all the 5 cases (6 years or below) in whom prick test to food allergen was performed in the present study, showed negative skin test.

POLLENS IN RESPIRATORY ALLERGY

Six cases (21.43%) in the present study gave positive reaction to pollens. There were only 4 pollens

extracts viz. Holoplelia, Parthenium, Pennisotum and Triticum used in the present study. Positive reaction to pollen of Parthenium hysterophorus was the commonest, among pollens, in the present study 17.85% cases - all having bronchial allergy. Out of 6 pollens sensitive cases, 4 gave positive reaction to Parthenium hysterophorus. This can be explained on the basis of high growing of Parthenium in Jhansi, even in the medical college campus. In the study of Pherwani et al (1985) pollens of Amaranthus spinosus and cocus nuciferus were the most common allergens giving positive reaction followed by Parthenium possibly because common pollens found in Bombay were of Amaranthoid group.

Jha et al (1975) showed highest allergenicity (33%) with Ageratum, among pollens, in bronchial allergy followed by Putranjiva.

DUST IN BRONCHIAL ALLERGY

Table XXIV shows results of test for dust sensitivity in bronchial allergy cases, as reported by various researchers from several places of India.

Fourteen (50%) of bronchial allergy cases in the present study gave positive reaction to dust. Among various sources of dust, house dust, gave the highest positivity rate (19.3%). This could be the result of Jhansi having a dry climate for most of the time in a year. Other investigators (Shivpuri, 1971; Jha, 1975;

and Mittal, 1979) also reported positive results from house dust as being the most common among various dusts. Shanker et al (1979), however, reported highest positivity rate with cotton dust. Sethi et al (1986) reported highest positivity rate with rice dust, possibly because Raipur is a rice growing area. In the present study hay dust also showed significant positivity. Other investigators have not studied hay dust allergy.

The differences in sensitivity to different kinds of dust, shown by various workers could be explained on the basis of different ecological flora of Jhansi, Raipur, Delhi, Bombay and Varanasi. Among the various studies conducted so far, in India (mentioned above) results of present study are nearer to the study of Jha et al (1975) from Varanasi.

TABLE XXV : Incidence of seasonal variation and comparison with Jha et al (1975) and Gupta et al (1988).

Type of allergy	Percentage of seasonal variation		
	Present study	Jha et al (1975)	Gupta et al(1988)
Rhinitis	66.66	25.00	-
Asthma	76.47	43.90	56.60
Rhinitis with asthma	66.66	45.10	-

SEASONAL VARIATION IN NASOBRONCHIAL ALLERGY

In present study seasonal variation was present in most of the cases (Table XXV). Seasonal variation was present in 66.67% cases of rhinitis, 76.5% cases of asthma and 66.7% cases of rhinitis with asthma. Gupta et al

(1988) reported seasonal variation in 56.6% cases of asthma. Jha et al (1975) reported 43.9%, 45.1% and 25.0% cases of bronchial asthma, asthma with rhinitis and rhinitis cases respectively, depicting seasonal variation. In great majority of cases (80.7%) frequency of episodes increased during winter season.

Precipitating factors were present in 82.35% cases of asthma in the present study. List of specific precipitating factors was large and multiple in many cases (Table XVIII).

In majority of the patients (85.7%) cold was the precipitating factor, second in importance was ice cream (32.4%) and last was the viral fever in 10.7% cases. So, old dictim of avoiding cold, ice cream and Khatai in asthmatic children had a definite basis. Gupta et al (1988) observed that 56.6% cases of asthma had obvious precipitating factors present.

IMMUNOTHERAPY

Immunotherapy, desensitization or hyposensitization could be defined clinically, as the administration of offending allergens in increasing concentration in an attempt to ameliorate the symptoms associated with exposure to allergens. Immunologically, it is the immunologic manipulation of an organism in an attempt to induce a state of relative tolerance to a specific allergen. It was first defined by Bostock in 1819, and

later emperically employed for the treatment of grass (spring) catarrh by Noon in 1911.

Various studies support the efficacy of immunotherapy. Horman (1974), Metre et al (1980), Norman and Lichtenstein (1978), Aas (1971) have shown decreased bronchial sensitivity to dust extract in persons who had received immunotherapy against dust allergen. Warner et al (1978) reported that 50% of bronchial asthma patients after receiving immunotherapy, had resolution of late phase of bronchial reactivity.

In this study, many problems came up during immunotherapy. As an example, one case had repeated asthmatic attacks of asthma. Therefore, many times, immunotherapy was stopped and when interval was more than one week the dose given at the next injection had to be the same as previous one.

Some times a corticosteroid preparation was given for an acute attack of disease. In such situations also immunotherapy had to be stopped. Immunotherapy was restarted at least one week after the cessation of corticosteroid.

Yet, in another case painless gross hematuria occurred during immunotherapy. There was a history of minor fall from stairs, prior to haematuria. In that situation, case was admitted for observation and immunotherapy was stopped. Haematuria subsided 5 days after non-specific treatment viz. maintenance of fluid and

electrolyte balance, giving oral alkalisers etc. After 30 days of this episode immunotherapy was again started and no complaint occurred. This patient also had one attack of asthma in the beginning of immunotherapy.

Both local and systemic reactions could occur with immunotherapy. Forgacs et al (1968) reported a few cases of increased wheezing after injection of aqueous allergen extract. Shaeffer et al (1984) showed that hyposensitization was quite safe but severe life threatening anaphylaxis could be expected though these rarely occurred. In the present study no adverse reaction occurred during immunotherapy of two patients.

In the earliest controlled trial (Brunn, 1949) 78% of the patients treated with house dust extract improved or were free from symptoms compared with 29% of the control group. But Mc-Allen (1961) found that house dust extract hyposensitization was ineffective, while treatment by inhalations of an aerosol of house dust extract gave good though short lived results.

British Tuberculosis Association (1968) observed that neither the treatment with house dust extract nor advice on dust control was advantagenous. The explanation given simply was that the treatment of asthma by this method was ineffective or that the effect was so small that it could not be demonstrated or else there was a low concentration of mite antigen used in the extract.

Bruce (1971) in his study of ragweed pollen observed no significant improvement after immunotherapy.

In present study immunotherapy was done in two patients of bronchial asthma (Table XX). In one patient, no improvement was noted after the initial treatment of immunotherapy, either in symptomatology or in peak expiratory flow rate. In other case, at the near end of initial treatment, symptomatology and peak expiratory flow had improved.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The present study was conducted at the Allergy Clinic, Immunology and Biochemistry laboratory, Department of Pediatrics, M.L.B. Medical College, and Hospital, Jhansi during the period of 14 months from October, 1991 to November, 1992. A sample of 57 children which included 48 allergy patients and 9 children as control cases were considered in the study. All the cases were below 21 years of age. The study group was divided into following categories of patients - Bronchial asthma (34), allergic rhinitis (3), urticaria (8), and allergic rhinitis with bronchial asthma (3).

Primary aim of present study was to study the spectrum of allergic illness in children, and to confirm their allergic nature, by skin prick testing, using 21 allergens.

The reactivity of histamine at different ages was noted and precipitating factors in allergic disorders were observed. Furthermore, the effectiveness of immunotherapy in the asthmatic children was studied.

Diagnosis was based on detailed history, clinical examination and relevant investigations. Emphasis was given to frequency of attacks in a year, precipitating factors, history of worm infestation and family history of allergy.

Besides skin prick test, hemoglobin, total leucocyte count, differential leucocyte count and erythrocytes sedimentation rate were done. Absolute eosinophil count was calculated and stool examination was done for ova/cyst.

Allergy testing was done by modified prick test method using prick test solutions. Saline was used for negative control and histamine for positive control.

Immunotherapy was done by commercially available aqueous allergen extracts, to hyposensitize those patients who were diagnosed as being sensitive to allergen (after skin prick testing).

FAMILY HISTORY OF ALLERGY AND SEX

In the present study positive family history of atopy was found in 35.3%, 66.7%, 12.5% and 33.3% cases of bronchial asthma, allergic rhinitis, urticaria and bronchial asthma in conjunction with allergic rhinitis respectively. Present history found a family in which one parent (father) was atopic and his two children (one male and one female) were atopic out of a total 3 children he had. In the present study it was observed that naso-bronchial allergy was common in male as compared to female (3 : 1) children. In urticaria, family history of atopy was present in 12.5% cases.

BLOOD COUNTS IN DIFFERENT CLINICAL GROUPS

All the patients and control cases had total

leucocyte count within the normal range. However, maximum mean eosinophil count of 8% was seen in bronchial asthma, allergic rhinitis and allergic rhinitis case associated with bronchial asthma.

Approximately 82.3% cases of asthma were having absolute eosinophil count more than 250/cmm, cases of allergic rhinitis had 66.6% and those of urticaria had 62.5%. Allergic rhinitis associated with asthma showed eosinophilia (AEC 7250/cmm) in 100% cases. In control group eosinophilia was seen in 11.5% cases. Eosinophilia was of the highest degree in those cases of bronchial asthma who had associated rhinitis (mean eosinophil count 805/cmm). In the present study a few cases of asthma (8.8%) had absolute eosinopenia.

INTESTINAL PARASITIS IN ALLERGIC DISORDERS

In bronchial asthma 4 out of 17 cases (23.5%) were positive on stool examination (two for cysts of Giardia lambia, one for Ankylostomata duodenale and one for cyst of ascaris lumbricoides). Absolute eosinophil count was higher in cases having helminthic infestation than in those who had protozoal infection or those who showed stool examination negative for ova/cyst.

Stool was positive for cysts of Giardia lambia in 20% urticaria cases and after the treatment for Giardia lambia, recurrence of urticaria did not stop.

SKIN PRICK TEST REACTION

Skin testing was done by modified prick method on volar surface of forearm. Histamine was used as a positive control and saline for negative control. In the present study both wheal and flare were measured by scale and divider.

Skin prick test was done in 25 cases out of 34 cases of bronchial asthma. Out of 25 cases 15(60%) showed positive reaction.

Skin prick test was done in three cases of allergic rhinitis and all cases showed positive reaction.

Skin prick test was done in three cases of bronchial asthma associated with rhinitis. Out of 3 cases two cases showed positive reaction.

Skin prick test was done in two cases of urticaria and one was positive.

REACTION OF HISTAMINE AT DIFFERENT AGES

Reaction of histamine was lowest in infants who were clinically suspected to be suffering from milk allergy. Among asthma cases maximum reactivity (both wheal and flare) of 30 mm (with multiple pseudopodia) was seen in an 8 years old child.

DUST IN BRONCHIAL ALLERGY

Fourteen (50%) of bronchial allergy cases gave positive reaction to dust. Among the various sources of

dust, house dust gave the maximum positivity rate(39.3%). This could be the result of Jhansi having a dry climate for most of the time in a year. The next common dust allergen was paper dust seen in 21.43% cases, followed by cotton dust in 17.86% cases.

POLLENS IN BRONCHIAL ALLERGY

Six cases (21.43%) in the present study gave positive reaction to pollens. There were only 4 pollen extracts viz. Holoptelia, Parthenium, Pennisetum and Triticum used in the present study. Positive reaction to pollen of Parthenium was commonest (4 our of 6 cases) among pollens. This could be explained on the basis of high growing of Parthenium in Jhansi.

INSECTS IN RESPIRATORY/BRONCHIAL ALLERGY

Eight cases (28.5%) gave positive reaction to insect antigens. In the present study two insect antigens viz. mosquito and D.farinae were used. Positive reaction to mite (D. farinae) was the commonest among insect allergens (14.28%).

FUNGUS IN RESPIRATORY ALLERGY

Two cases (7.14%) gave positive reaction to fungus i.e. Aspergillus flavus, the only fungal antigen in the present study.

SEASONAL VARIATION IN NASOBRONCHIAL ALLERGY

Seasonal variation was present in 66.7% cases of rhinitis, 76.5% cases of asthma and 66.7% cases of rhinitis with asthma. In 80.7% cases frequency of episodes increased during winter season.

PRECIPITATING FACTORS IN NASOBRONCHIAL ALLERGY

Cold was the precipitating factors in 85.7% cases. Second in importance was ice-cream(32.4%) and last was the viral fever in 10.7% cases. So, old dictim of avoiding cold, ice-cream and khatai in asthmatic children might have a definite basis.

SKIN PRICK TEST IN CHRONIC URTICARIA

Skin prick test was done in two cases and one was positive for urad dal. But, on withdrawal of urad dal, symptoms did not subside.

IMMUNOTHERAPY IN BRONCHIAL ASTHMA

In the present study immunotherapy was done in two cases. One case suffered from recurrent asthmatic attacks during hyposensitization and no beneficial effect was observed. While in other case of hyposensitization at the end of initial treatment, amelioration of symptoms and increase in peak expiratory flow rate was observed.

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A P P E N D I X

APPENDIX

WORKING PROFORMA

EVALUATION OF PRICK TEST IN VARIOUS ALLERGIC DISORDERS.

Place of work : Allergy Clinic, Immunology and Biochemical Laboratory, Department of Pediatrics, M.L.B. Medical College, Hospital, Jhansi.

Case No. _____

OPD No. : Data :

Code No. :

Name : Age/Sex :

Father's name :

Address :

Diagnosis :

Occupation of Father : Mother :

Total family Income : Rs. /month.

Per capita Income : Rs. /month.

Geniological History:

Social : Residence.

Vegetarian/Non vegetarian(Pork, cow) :

Method of excreta disposal :

Immunization	Primary	Booster
Polio		
D.P.T.		
B.C.G.		
Measles		
Others		

HISTORY OF PRESENT ILLNESS

Symptoms	Duration
1. Fever	
2. Cough	
3. Breathlessness	
4. Wheezing	
5. Excessive cry	
6. Abdominal colic	
7. Irritability	
8. Loose motion	with blood.
9. Rashes : Dermatitis	
Urticaria	

Related to Ingestants: (Fish, Shellfish, nuts, peanuts, food additive, drugs etc.).

- Yes/No
- Specific Name
- Average time interval - Days
  Hours

Related to Contactants : (Plant substances drugs applied to the skin stinging nettle, animal saliva etc.)

- Yes/No
- Specific Name
- Average time interval - Days
  Hours

Related to Inhalants (Pollen, dander, ? molds etc.)

- Yes/No
- Specific Name
- Average time interval - Days
  Hours.

Related to Infectants : (Drugs, transfused blood, therapeutic antisera, insect stings and bites, allergenic extracts etc.)

- Yes/No
- Specific Name
- Average time interval - Days
  hours

Related to Parasitic intestinal infestations :(Passing worms, vague abdominal pain, PICA, perianal itching, failure to thrive, acute abdomen motions/Constipation).

Related to other Parasitic infections/Infestations

- Malaria

Filaria

- Scabies
- Others

Causative/Precipitating factors:(Time interval between exposure and symptoms

- Cold
- Solar
- Aquagenic
- Vibratory
- Dermagraphism
- Physical Exercise.
- Emotional stimuli
- Parasitic infection
- Viral Infection
- Bacterial infection
- Fungal infection.

Details of Present illness

1. Duration of symptoms/episode
2. Frequency per year
3. Seasonal variation, if any
   (Note : specify the season)

HISTORY OF PAST ILLNESS

Note :(Specify only the duration)

- Measles
- Pertussis
- Chronic diarrhoea
- Worm infestations
- Others
- Urticaria
- Eczema
- Asthma
- Rhinitis

FAMILY HISTORY (Details if any)

- Koch's lung : Yes/No
- Chronic illness : Yes/No
- Similar illness : Yes/No

HISTORY OF ATOPY : (Asthma, urticaria, atopic dermatitis, Rhinitis, history of angioedema, fever).

PHYSICAL EXAMINATION

H.R./P.R.
Resp. rate
Temperature
Blood pressure
Pallor
Ecterus
Cyanosis
Clubbing
Hydration

Edema : Yes/No
Lymphadenopathy
- Cervical
- Axillary
- Others
Weight
Head circumference
Height/Length
Peak expiratory flow

Nutrition

Examination of skin

Wheal

Size

Distribution

Scalp

- Face
- Neck
- Trunk
- Extremities : Upper - Flexor surface
  Extensor surface
  Lower - Flexor surface
  Extensor surface

Dermatitis

A. Distribution
   - Scalp
   - Face
   - Neck

- Trunk
- Extremities : Upper - Flexor surface
  extensor surface
  Lower - Flexor surface
  Extensor surface

B. Nature of lesion (Groups papules, vesicles, oozing, scaling, mention other features, Erythma, Excoriation).

SYSTEMIC EXAMINATION

Respiratory system

Inspection

Palpation

Percussion

Auscultation

Cardiovascular system

Signs of CHF

J.V.P.

H.J.R.

Hepatomegaly

Basal crepts

Abdomen

- Spleen
- Liver
- Colon
- Ileocaecal lump
- Tenderness
- Lymphnode

Central Nervous System

INVESTIGATIONS

Blood : T.L.C. cells/cu.mm.
D.L.C.: P %, L %, E %, M %, B %
Hb gm%, ESR : mm in 1st hour
Absolute eosinophil count :

Stool : Ova/Cyst RBC/Pus cells.

Others: X-ray chest, Mantoux test etc.

SKIN TEST

Name	Date	Result	Remarks

1. Specific allergen test by modified Prick Method
   a.
   b.
   c.
   d.
   e.
   f.
   g.
   h.
   i.
   j.